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POSTOPERATIVE SINUS THROMBOSIS

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CHICAGO

In 1929 I reported 154 cases of otitic sinus thrombosis with a mortality rate of 22 per cent. Among these cases were 22 in which the classic symptoms of sinus thrombosis were not manifested until after the performance of the mastoid operation. An exhaustive study of these 22 cases revealed that they included two well defined types of sinus thrombosis, namely, latent and postoperative.[1]

Since the time of that communication I have observed other cases of latent and of postoperative sinus thrombosis which in general confirmed the description given in the original paper. I believe that the distinction between the two types of sinus thrombosis is of such clinical importance that I should take the opportunity to present 2 cases recently observed at the Research and Educational Hospital of the University of Illinois, which afford excellent examples of the two clinical types of the condition.

REPORT OF CASES

CASE 1.—*Latent Sinus Thrombosis.*

R. S., a Negro 18 years old, had had measles when he was 6 years old and, as a sequel of the measles, otitis media on the right, which responded favorably to conservative measures. Two weeks before admission to the Research and Educational Hospital he caught a cold, and fever developed. One week after this onset the right ear began to discharge. There was continual fever with temperatures as high as 101 F. On June 10, 1939 it rose to 105 F. and the patient had a headache and a chill. On admission the right ear evidenced a scanty suppuration, redness of the drum and mastoiditis. On June 12 a simple mastoid operation was performed. The mastoid process was found to be diploetic and the antrum narrow, but no pus was found in it. The bony plate overlying the lateral sinus was hard and intact. Nevertheless, it was deemed advisable to expose the sinus and the dura of the middle fossa. The wall of the sinus was normal.

Immediately after the operation fever occurred (fig. 1). Hemolytic streptococci were found in the blood as well as in the culture from the mastoid process, so

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1. Brunner, H.: Postoperative Sinus Thrombosis, Arch. Otolaryng. **10**:217 (Sept.) 1929.

that the patient was given sulfanilamide. On June 25 the left wrist was swollen; on the following day an enlargement of the spleen was discovered, and then on the next day there were metastases in the left wrist, the left knee and the right shoulder. On June 28 the jugular vein was ligated, and the mastoid wound was reopened. The sinus was covered by normal granulations and was exposed to a point close to the torcular Herophili. After an incision of the sinus was made, frank pus flowed out, but there was no free bleeding, either from the torcular Herophili or from the jugular bulb. A small curet was introduced into the torcular Herophili as well as into the jugular bulb, and thrombi were removed. Free bleeding then occurred from both ends. In the after-treatment much pus drained from the region of the jugular bulb. On July 19 the boy was dismissed as recovered from the sinus thrombosis as well as from the metastases.

Laboratory Findings.—The results of examination of the blood are shown in the following tabulation:

	June 12	June 18	June 25	June 28
Erythrocytes	4,030,000	.....	3,090,000	4,050,000
Leukocytes	14,090	10,020	8,090	14,000
Polymorphonuclear leukocytes	85%	70%	60%	80%
Lymphocytes	10%	20%	30%	10%
Monocytes	5%	10%	10%	10%

The icterus index on June 13 was 10, on June 21 was 8, on June 29 was 5 and on July 11 was 5.

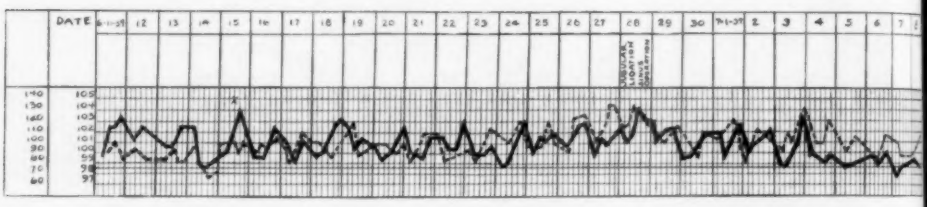

Fig. 1.—The temperature curve in a case of latent sinus thrombosis; *X*, *Komplikationssacke.*

There are certain clinical features which render this case a typical case of latent sinus thrombosis.

1. The definite signs of sinus thrombosis (high grade fever, chills, metastases) appear only after the mastoid operation. Before the mastoid operation the sinus thrombosis is more or less latent. Admittedly the term "latent" is misleading. Too often "latency" is just another expression of insufficient experience or incomplete observation on the part of the physician, although it sometimes happens that a sinus thrombosis runs a course even without fever. The condition of R. S. was by no means "latent." The continued fever, the scanty discharge, the headache and the chill prior to the mastoid operation indicated definitely a complication of mastoiditis. This was the reason for exposing the sinus and the dura at the operation on the mastoid. Nevertheless, the symptoms were not so marked as to allow the definite diagnosis of sinus thrombosis and to indicate the operation on the sinus and the jugular

vein. It is for this reason that these conditions should be termed "latent sinus thrombosis," although there is no latency in the actual sense of the word.[2]

2. Latent sinus thrombosis occurs more frequently in cases of acute suppurative otitis or acute recurrent otitis than in cases of chronic otitis. In the case of R. S. there was acute recurrent otitis. A diploetic appearance of the mastoid process and rapid development of a septic state are frequently found in these cases, particularly if the patients are young.

3. The mastoid operation revealed but little suppuration and not much destruction of bone but a marked hyperemia; these conditions correspond to the findings in the osteophlebitis pyemia of Koerner or in the hemorrhagic mastoiditis of Kopetzky. In some cases of latent sinus thrombosis granulations are to be found in the antrum. In the case of R. S. the bony plate of the sinus was firm, and the wall of the sinus between the superior and the inferior knee was normal.

4. On the day after the operation (in other cases, on the day of the operation) the temperature rose sharply (*Komplikationszacke*). Chills may or may not accompany such a rise of temperature.

5. When the sinal operation is performed a short time after the mastoid operation (two to four days), the wall of the sinus is found to be covered by granulations, which, however, are neither necrotic nor pinkish red. On the contrary, they often appear as normal granulations, which are found in every healing mastoid cavity. Consequently, it is amazing to find a great amount of frank pus and thrombi within the sinus itself. In this manifestation latent sinus thrombosis differs markedly from the osteophlebitis pyemia of Koerner, in which a marked pathologic change of the sinus is as a rule absent. In the case reported here the sinal operation was delayed until sixteen days after the original mastoid operation, in the hope that sulfanilamide therapy would be effective. However, the conditions seen at the operation did not differ from those which can be discovered when operative intervention is employed earlier in the course.

6. Provided that the sinal operation is performed in the proper manner, the sepsis gradually decreases, and in the majority of the cases there is recovery. As I have previously mentioned, sulfanilamide did not influence the course of the latent sinus thrombosis in the case of R. S.

7. When latent sinus thrombosis produces metastases, they are usually localized in the joints, less frequently in the lungs. This corresponds to the observations of Koerner in cases of osteophlebitis pyemia.

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2. Compare Lederer, F. L.: Pathogenesis and End-Results of Dural Sinus Thrombosis, Arch. Otolaryng. **19**:410 (March) 1934.

Considering these clinical features, it is obvious that latent sinus thrombosis is not postoperative sinus thrombosis, although the definite symptoms of the condition appear after the mastoid operation; it does not develop as a new process but becomes manifest only after the operation.

The results of exact microscopic examination of temporal bones in cases of latent sinus thrombosis are not available, as the majority of patients with this condition recover. Consequently, the pathologic picture in these cases is, to a great extent, hypothetic. Nevertheless, there cannot be doubt that sinus thrombosis of this type develops by the endophlebitic and not by the periphlebitic route. As the result of often repeated microscopic examinations it is well known that the classic sinus thrombosis develops from an infectious process adjacent to the wall of the sinus. At first the infection invades the connective tissue of the wall of the sinus, producing sinal phlebitis. When it progresses it destroys the endothelial layer of the sinus, thus preparing for the development of a thrombus. This constitutes the periphlebitic route.

In cases of latent sinus thrombosis there is no collection of infectious material adjacent to the wall of the sinus, and there is no macroscopic inflammation of the connective tissue of the sinus. In these cases the infection begins rather in the venous channels which are connected with the sigmoid sinus. There are various channels of this type which come into consideration. I mention the emissary veins, the little veins within the mastoid process, and the jugular bulb. In all these channels a thrombus may develop in the usual periphlebitic route, because of an adjacent collection of infectious material. From these channels the thrombophlebitis may extend into the sigmoid sinus, producing sinus thrombosis. Consequently, in these cases the sinus thrombosis does not develop from outside to within, but from within outward, that is, by the endophlebitic route. This is what occurs in cases of latent sinus thrombosis.

Actual postoperative sinus thrombosis is characterized by different clinical symptoms and by a different clinical course. The following case is an example.

CASE 2.—*Postoperative Sinus Thrombosis.*[3]

J. C., a white woman 33 years old, in 1932 noticed for the first time a tumor blocking the right external auditory canal. The tumor was removed in 1934. In 1937 there was a recurrence. The patient did not complain of earache except when she caught a cold.

3. This type of sinus thrombosis has, of course, nothing to do with the postoperative sinus thrombosis occurring after operations on the abdomen, chest and other regions, which most frequently is found in children and affects principally the superior longitudinal sinus. (Evans, P. R.: Postoperative Intracranial Thrombosis in Childhood, Lancet **2**:12, 1935.)

On admission (Nov. 27, 1939) there was found a red firm tumor blocking the entire right external auditory canal. Microscopically the tumor proved to be an angiofibroma. There was a great nevus occupying the right half of the neck. On November 28 a conservative radical operation was performed, and the tumor was removed. The sigmoid sinus was exposed to a small extent, but the exposed sinus was not connected with the mastoid cavity, as will be brought out later in this report. In the postoperative treatment there was much fetid discharge. Nevertheless, the postauricular wound closed on December 10. Two days later the patient complained of dizziness. When the dressing was being changed, the patient fainted and became cyanotic. General convulsions of tonic-clonic type were noticed; the eyes deviated upward and to the right. The patient was given sulfanilamide and azosulfamide.[4] In the evening she felt well. There were no neurologic or meningeal symptoms, and there were no chills. On December 13 there were two chills, and the temperature rose to 106.8 F. (fig. 2). There was a convergence spasm of the eyes, paresis of the external rectus muscle on the left and vomiting but no other meningeal symptoms. Spinal puncture revealed a pressure of 50 mm. of water, which increased to 80 mm. after pressure on the jugular vein. The spinal fluid contained 65 mg. of dextrose per hundred cubic centimeters and a slight amount of protein, but no bacteria were found. The

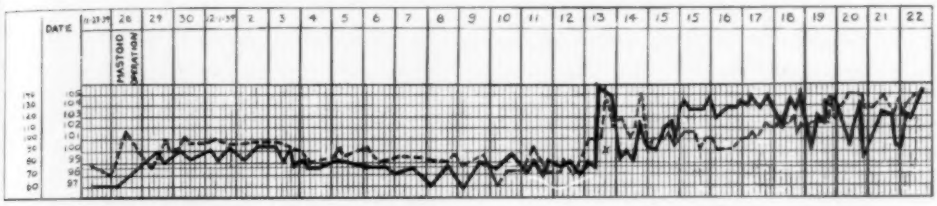


Fig. 2.—The temperature curve in a case of postoperative sinus thrombosis; *X*, *Komplikationszacke*.

wound was reopened. The previously exposed wall of the sinus, covered by healed over skin, had no connection with the mastoid cavity. At that site the wall of the sinus was somewhat necrotic and was partly covered by granulations. The sinus was exposed to a larger extent than in the earlier procedure. The other portion of the sinal wall was white and thick but smooth. The tip of the mastoid process and the lateral part of the floor of the bony external canal were removed, as the bone was infiltrated by granulations. The lateral wall of the attic was removed, together with the malleus, which was found to be infiltrated by granulations. The sinus was incised. Free bleeding occurred, which was stopped by a tampon. The jugular vein was not ligated because of the absence of an obturating thrombus within the sinus, the poor general condition of the patient and the nevus at the right side of the neck. On December 15 the fundi of the eyes were found to be normal. There were no meningeal symptoms. The cerebrospinal fluid was clear and sterile. On December 16 the spinal fluid was under extremely low pressure, so that it had to be aspirated. It was then found to be cloudy. There were gram-negative cocci in the spinal fluid, intracellularly and extracellularly situated.

4. Azosulfamide is disodium 4-sulfamidophenyl-2′-azo-7′-acetylamino-1′-hydroxy-naphthalene-3′,6′-disulfate. This substance has been known as prontosil soluble, as prontosil and as neoprontosil.

No meningeal symptoms were present and no choked disks were found. On December 18 the patient complained of pains in the chest, which were due to metastatic pneumonia. On December 20 jaundice was noticed, and on December 22 the patient died.

Laboratory Findings.—The results of examination of the blood are shown in the following tabulation:

	Nov. 27	Dec. 12	Dec. 17	Dec. 20
Erythrocytes	4,200,000	3,700,000	4,300,000	4,200,000
Leukocytes	7,200	10,000	15,300	20,100
Polymorphonuclear leukocytes	70%	.....	84%	.....
Lymphocytes	26%	.....	12%	.....
Monocytes	4%	.....	2%	.....
Juvenile forms	.....	.....	2%	.....

Autopsy.—The autopsy revealed the conditions typical of otitic sepsis, but there was no evidence of meningitis. Other observations made at autopsy, which have no connection with the subject under discussion, will be dealt with at another time.

This is a typical case of postoperative sinus thrombosis and differs definitely from the case of R. S., which was diagnosed as one of latent sinus thrombosis.

1. Signs of sinus thrombosis appear long after the mastoid operation. Before the mastoid operation the signs of sinus thrombosis or sepsis are definitely absent. In latent sinus thrombosis preoperative symptoms of a complication are present as a rule, although they do not allow for a certain diagnosis.

2. Postoperative sinus thrombosis occurs more often in cases of chronic otitis. In the case of J. C. there was chronic otitis combined with a tumor, probably originating in the external auditory canal. Latent sinus thrombosis occurs more frequently in cases of acute suppurative otitis.

3. At the mastoid operation the conditions typical of chronic otitis were discovered, but there was no extension of the suppuration toward the sinus, and the wall of the sinus was normal. In latent sinus thrombosis the symptoms typical of acute mastoiditis, often of the hemorrhagic type, are present.

4. Immediately after the mastoid operation the temperature was normal and stayed normal. In the case of J. C., fifteen days after the mastoid operation a sudden rise of the temperature (*Komplikationszacke*) occurred, which was followed by a typical septic temperature. In latent sinus thrombosis the *Komplikationszacke* appears immediately after the operation.

5. When the sinal operation was performed, but few changes on the wall of the sinus were found. The wall of the sinus, which was exposed at the original mastoid operation, was covered by granulations, which appeared dark and necrotic. However, it must be emphasized that the prior exposure of the wall of the sinus is not of prime importance for

the development of postoperative sinus thrombosis. In cases of latent sinus thrombosis marked changes within the sinus itself are found.

6. Although the sinal operation was performed in the proper way, the disease ran a fulminant downhill course. That is the rule in cases of postoperative sinus thrombosis. Ligation of the jugular vein and the administration of sulfanilamide and azosulfamide usually have no specific effect.

7. When postoperative sinus thrombosis produces metastases they are, as a rule, localized to the lungs (abscess of the lung, empyema or pneumonia) or to the brain (meningitis, purulent encephalitis) and less frequently to the joints. Latent sinus thrombosis most frequently produces metastases to the joints.

COMMENT

As Lillie [5] has pointed out, postoperative sinus thrombosis may occur when the surgeon has incompletely removed the diseased tissue at the time of the original operation. That may occur particularly in instances in which the simple mastoid operation has been done in the treatment of acute otitis and pneumatic cells left in the mastoid process. In my experience postoperative meningitis usually develops in such cases rather than postoperative sinus thrombosis, which will be found more often after radical mastoid operations, as I have mentioned before. Consequently, I cannot quite agree with Lillie, who pointed out that signs of otitic sepsis which occur late after a mastoid operation merely indicate "that the pathologic process has not been controlled by the original operation." I rather believe that these signs occasionally occur even when the pathologic process has been controlled by the original operation. The reason for the development of postoperative sinus thrombosis in such cases is usually a secondary infection of the wound.

There is no reason to contest the relative importance of an infection of the wound for the development of postoperative sinus thrombosis, particularly when such an infection is combined with retention of an inflammatory exudate. In such cases, it is not of prime importance that the wall of the sinus was exposed at the time of the original operation, although unquestionably the exposure of the sinal wall favors the development of postoperative sinus thrombosis. More dangerous than the mere exposure of the wall of the sinus is the unintentional production of a lesion of the wall, inasmuch as the quick and forcible tamponade in these cases favors the development of a secondary infection.

This concept explains well the two most prominent characteristics of postoperative sinus thrombosis. Inasmuch as in these cases an acute and severe infection, originating in the mastoid cavity and not in the middle ear, invades and rapidly penetrates the wall of the sinus, there

5. Lillie, H. J.: The Surgery for Sepsis of Otitic Origin, in Kopetzky, S. J.: Surgery of the Ear, New York, Thomas Nelson & Sons, 1938, p. 26.

is, as a rule, insufficient time to mobilize the defense forces of the sinal wall and of the blood stream. Consequently, one misses the extensive periphlebitic thickening of the wall as well as the development of large thrombi within the sinus. On the contrary, instead of producing a thickened sinal wall the infection leads occasionally to a thinning and even a necrosis of the wall, which may be followed by a spontaneous rupture and a profuse hemorrhage. Such cases I have mentioned in my first paper.[1]

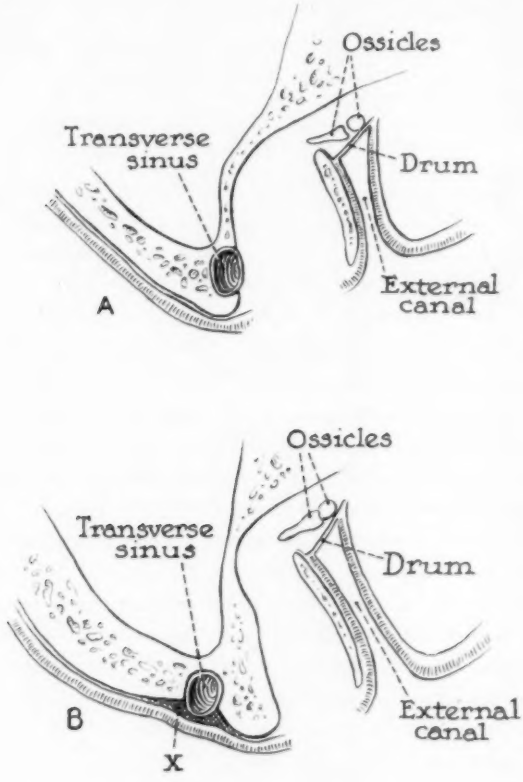


Fig. 3.—*A*, horizontal section through the external auditory canal, the antrum, the mastoid cavity and the sinus, showing the correct way of exposure of the sinus. *B*, horizontal section through the external auditory canal, the antrum, the mastoid cavity and the sinus, showing the incorrect way of exposure of the sinus. *X*, pocket between the skin and the sinus.

In the case of J. C. the pathogenesis of the postoperative sinus thrombosis is of particular interest. The mastoid operation was performed by a young surgeon. The approach was begun too far occipitally, and consequently the sinus was immediately exposed (fig. 3 *B*). The most important error, however, occurred in the covering over of the exposed

wall of the sinus with skin and in starting a new operative terrain closer to the posterior wall of the external auditory meatus. Thus conditions were created which made possible the development of postoperative sinus thrombosis. The proper procedure is to drain the area of the exposed wall of the sinus into the mastoid cavity, as indicated in figure 3 *A*. When the exposed wall of the sinus was covered with skin, infectious material accumulated within a pocket (*x* in fig. 3 *B*), thus producing an infection of the exposed wall of the sinus. Similar conditions are encountered when a surgeon, in performing a radical mastoid operation, cuts a skin flap from the posterior wall of the external auditory canal and covers the exposed wall of the sinus with the flap. It is well known that such a procedure may lead to the development of postoperative sinus thrombosis.

The described concept of differentiation between latent and postoperative sinus thrombosis was accepted in general by Kriegsmann,[6] Meltzer[7] and Ersner.[8] The subject was discussed in an excellent manner by Boies,[9] who reported 184 cases of lateral sinus thrombosis with a mortality rate of 25 per cent. As far as postoperative sinus thrombosis is concerned, Boies generally expressed agreement with my concept. However, the mortality rate in cases of postoperative sinus thrombosis reported by him was much lower, namely, 15.7 per cent, compared with the mortality rate in the 9 cases of my previous paper, which was 67 per cent (not 50 per cent, as Boies quoted). Intending to explain this conspicuous difference, Boies expressed the belief that in my cases frequently only an "antrotomy" was performed, without removal of all inflamed cells. That statement suggests a misunderstanding of the term "antrotomy" as I employ it. In all of my years in the field of otolaryngology I have never performed an "antrotomy" or seen one performed in the otolaryngological department of the Policlinic in Vienna. The term "antrotomy" was used to designate the simple mastoid operation, which, however, is not always an actual mastoidectomy. This also holds true for the modern concept of simple mastoid operation. It is necessary only to make routine and thorough roentgen examinations of the patients on whom the simple mastoid operation is performed according to the modern concept, and one is surprised how often it is that even under these circumstances pneumatic cells

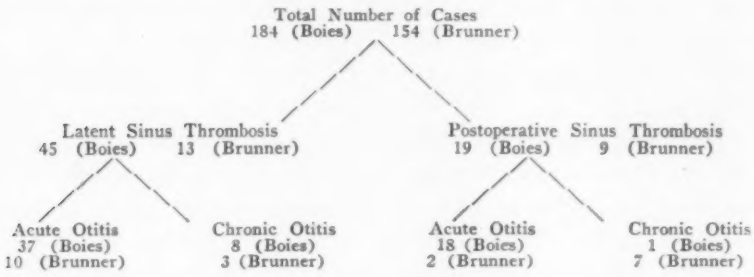
6. Kriegsmann: Zur Frage der postoperative Sinusthrombose, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **131**:164, 1937.

7. Meltzer, P. E.: Thrombosis of Lateral Sinus, Arch. Otolaryng. **22**:131 (Aug.) 1935.

8. Ersner: Diagnosis of Otitic Surgical Lesions, in Kopetzky, S. J.: Surgery of the Ear, New York, Thomas Nelson & Sons, 1938, p. 21.

9. Boies, L. R.: Lateral Sinus Thrombosis, Ann. Otol., Rhin. & Laryng. **41**:227, 1932.

are left within the temporal bone. There is no doubt that the same thing happened in the past, when the simple mastoid operation was more frequently performed and was discussed less. Therefore I do not believe in condemning the technic of the simple mastoid operation as the chief source of the difference between the mortality rate reported by Boies and the mortality rate in my cases. I particularly do not stress this point as the most important, inasmuch as it has been my experience that actual postoperative sinus thrombosis occurs more frequently after radical operations, which usually do not concern a well pneumatized mastoid process. Consequently, there must be other grounds on which to account for the difference between the findings of Boies and those reported in my series. It is possible that Boies applied the term "postoperative sinus thrombosis" to other conditions. Such a suspicion first arises when one places the corresponding numbers of cases observed by Boies and by me in one table. The total of my cases differs somewhat from the numbers which Boies quoted from my previous contribution. However, my previous paper did not deal with statistics. Its chief purpose was the description of the clinical picture of latent and of postoperative sinus thrombosis as exemplified by typical cases.



There are such great differences between the different groups of cases that the disparity scarcely can be explained as due to incidental differences of observation. This particularly holds true so far as the cases of postoperative sinus thrombosis are concerned. Such differences can be explained only by fundamental differences of the clinical interpretation of the cases. Unfortunately Boies described but 3 cases of postoperative sinus thrombosis exhaustively. Although it is not entirely possible to estimate a case from the description of the clinical course, without having observed the patient, I have reasons to believe that among the 3 cases of Boies, only 1 (case 3) resembles the picture of postoperative sinus thrombosis. In Boies's second case there was partial destruction of the sinal plate and there were granulations on the sinus at the time of the original mastoid operation; in his case 4 there was a sudden cessation of discharge followed by earache, mastoiditis and three or four chills prior to the mastoid operation. Therefore, in both cases the sinus was

apparently not normal before the mastoid operation. In my previous paper I stated, and I again reiterate, that a normal condition of the sinus before the mastoid operation is one of the chief requirements for the diagnosis of postoperative sinus thrombosis.

Latent and postoperative sinus thrombosis are two distinct types of sinus thrombosis, each with a certain fundamental pathologic character, symptom complex and prognosis. The description of morbid entities can scarcely avoid being more or less schematic. Nevertheless, the knowledge of these types is necessary to enable the clinician to discover what is the accepted rule and what is the exception. Undoubtedly, exceptions to the typical pictures presented will be found when knowledge concerning latent and postoperative sinus thrombosis increases, as the result of more careful clinical observation.

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LARYNGOFISSURE FOR CANCER OF THE LARYNX

OBSERVATIONS BASED ON A SERIES OF FIFTY CONSECUTIVE CASES

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"Laryngofissure" means simply splitting of the larynx, and for this reason some object to the application of the term to the operation for removal of a part of the larynx when it is affected by cancer in an early stage. Martin,[1] for example, prefers to call all such operations "partial laryngectomies." Another objection to the former term is that the word implies splitting of the whole larynx, and for this reason the term "thyrotomy" has been used. The objection to the latter term, that it could be taken to refer to the thyroid gland, led to the use of "thyrochondrotomy." "Laryngotomy" was the term used by Desault in 1812, when he first described the operation. Throughout this paper, as in Moore's excellent monograph,[2] "laryngofissure" is used "in its old comprehensive sense as including both the longer and the shorter incisions.

Whereas Desault (1812) had suggested this operation for the removal of impacted foreign bodies rather than for the removal of growths, Brauers, of Louvain, Belgium, first performed a laryngofissure in order to obtain access to a growth. He cauterized a papillomatous tumor in a man 40 years of age, and the patient survived for twenty years. Gurdon Buck, of New York, was the first to do the operation for removal of a malignant growth (1851). In the next few years, an increasing number of attempts were made to utilize the laryngofissure approach, a considerable impetus being given by the discovery and development of laryngoscopy (1855). However, the results were very unsatisfactory, as shown by von Bruns (Berlin, Germany, 1878), who reported a series of 19 cases, in which only 2 patients survived one year! The British laryngologists, including Morrell, Mackenzie and even Butlin and Semon, later outstanding exponents of laryngofissure, joined in condemning the operation. The bad results were due to use of the procedure

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Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society, Inc., in 1940.

1. Martin, H. E.: The Treatment of Cancer of the Larynx, Tr. Am. Acad. Ophth. **41**:213-268, 1936.

2. Moore, I.: Intrinsic Cancer of the Larynx and the Operation of Laryngo-Fissure, J. Laryng., Rhin. & Otol. **33**:129 (May); 166 (June); 202 (July); 234 (Aug.); 260 (Sept.); 294 (Oct.) 1918.

in cases in which the growth had reached an advanced stage and conditions were unsuitable for the operation, as well as to faulty technic. After 1890, both Butlin and Semon, working first together and later separately, steadily improved their results, and, as Moore stated, "owing to the diagnostic skill of Semon and the operative skill and technic of Butlin," this period was "one in which great advances were made, resulting in this operation being reinstated in its proper place." Other pioneers whose statistics began to show what could be done by the use of laryngofissure in properly selected cases were Schmiegelow, Chiari, Moure, Delavan, St. Clair Thomson and Chevalier Jackson.

For full bibliographies, in which reference to all of the early writers mentioned can be quickly found, see Irwin Moore's monograph,[2] St. Clair Thomson and Colledge [3] or the book of Jackson and Jackson on cancer of the larynx.[4c]

TECHNIC

I shall refer first to the technic I have used in the series of cases analyzed in the accompanying table and then give some historical and comparative data under the various successive headings.

Anesthesia.—Local anesthesia was used in all my cases. It was induced by infiltration of 1 per cent procaine hydrochloride injected intradermally and then subcutaneously along the midline of the neck. It was completed by topical application to the interior of the larynx and upper part of the trachea of cocaine (10 per cent) on a swab introduced through an incision in the cricothyroid membrane, as will be described subsequently. This application is analgesic, eliminates the cough reflex from the larynx and upper part of the trachea while conserving it in the lower part of the trachea and the bronchi and produces slight ischemia of the mucous membrane and soft tissues of the interior of the larynx, lessening the tendency toward hemorrhage.

The first laryngofissures were done without anesthesia, general or local, and they certainly were a tribute to the courage of the patients and the skill and patience of the operators. Duncan Gibb was probably the first to do a successful laryngofissure for intrinsic cancer of the larynx in England, and he used chloroform (1864). The year before, Sands, of New York, had used ether for the same operation in a similar case. Unfortunately, both patients died later of recurrence. Chevalier Jackson

3. Thomson, St. C., and Colledge, L.: Cancer of the Larynx, New York, The Macmillan Company, 1930.

4. Jackson, C., and Jackson, C. L.: (*a*) The Larynx and Its Diseases, Philadelphia, W. B. Saunders Company, 1937; (*b*) Cancer of the Larynx, J. A. M. A. **111**:1986-1992 (Nov. 26) 1938; (*c*) Cancer of the Larynx, Philadelphia, W. B. Saunders Company, 1939.

for many years advocated intratracheal insufflation of ether but for the past ten or fifteen years has used local anesthesia exclusively, as have most other operators in this country.

Incision.—A straight vertical midline incision from just above the thyroid notch to just below the level of the cricoid was used in all my cases. If tracheotomy is to be done, the incision may be prolonged downward to the suprasternal notch. Loré [5] prefers to make a separate incision for the tracheotomy, leaving a bridge of skin between, as Solis-Cohen did some years ago. I did not do tracheotomy as a part of the original operation in any of the cases in my series, though the procedure was necessary in 2 cases in order to permit packing for the control of hemorrhage.

Chevalier Jackson has emphasized the importance of not skeletonizing the larynx in laryngofissure, because of the unnecessary trauma inflicted on the external perichondrium and the consequent impairment of nutrition of the thyroid alae.

Special Cover.—As soon as the incision has been made, the cartilages exposed and hemostasis accomplished, a special cover, consisting of a sheet with an opening in it just big enough to fit the wound, is put in place, the edges of the opening being clipped to the skin margins of the wound (fig. 1). This cover helps to maintain scrupulous cleanliness and to prevent contamination from the skin during the remainder of the operation, particularly during the manipulation of the cartilages.

Incision of Cricothyroid Membrane.—After placement of the special cover, the cricothyroid membrane is incised transversely or vertically for the topical application of cocaine to the interior of the larynx. At first a few drops are squeezed out by pressure of the sponge against the borders of the incision, and then the sponge itself is gently introduced.

Division of the Thyroid Cartilage.—In cases in which the lesion does not reach the anterior commissure (fig. 2 *A*), the "clipping" technic is used, the turbinotome being employed to divide the thyroid alae exactly in the midline (fig. 3), but in cases in which the lesion has reached the anterior commissure (fig. 2 *B*), the special technic for such cases first described by Chevalier Jackson [6] in 1922 is used (fig. 4). The essential feature of this technic is the division of the thyroid cartilage from the outside by means of a saw, or in the case of patients with a very soft

5. Loré, J. M.: Suggested Modifications of the Laryngofissure Operation, Ann. Otol., Rhin. & Laryng. **42**:1205-1223 (Dec.) 1933.

6. (*a*) Jackson, C.: The Results of Operative Methods in the Treatment of Cancer of the Larynx: Symposium, Proc. dixième Cong. internat. d'otol., Paris, 1922; Ann. d. mal. de l'orielle, du larynx **41**:1221-1239, 1922. (*b*) Jackson and Jackson.[4c]

cartilage, a knife, this incision not going clear through the internal perichondrium. In this way one avoids cutting through the tumor, which is always bad surgical procedure. In my series, the clipping technic was used in 32 cases and the Jackson "anterior commissure" technic in 18.

(In the tables, the clipping operation is designated as "A" and the Jackson anterior commissure operation as "B.")

Fig. 1.—The operation of laryngofissure. The thyroid cartilage has been exposed and the cricothyroid membrane incised. The next step is the topical application of an anesthetic to the larynx and the upper part of the trachea through the incision in the cricothyroid membrane. Then the thyroid cartilage is divided, either with shears ("clipping" technic) or with the circular saw ("anterior commissure" technic). Note the special cover attached to the margins of the skin.

Fig. 2.—Mirror appearance of lesions suitable for laryngofissure. *A*, a lesion involving the middle third of the cord, for which the clipping operation would be used. *B*, a lesion in the anterior commissure, which would require the use of the Chevalier Jackson anterior commissure technic (Jackson and Jackson [4b]).

Inspection of the Interior of the Larynx.—If the patient has been properly studied beforehand by direct as well as indirect laryngoscopic inspection, one should have an accurate idea of the location and extent of the lesion, but after the larynx is opened it should again be carefully

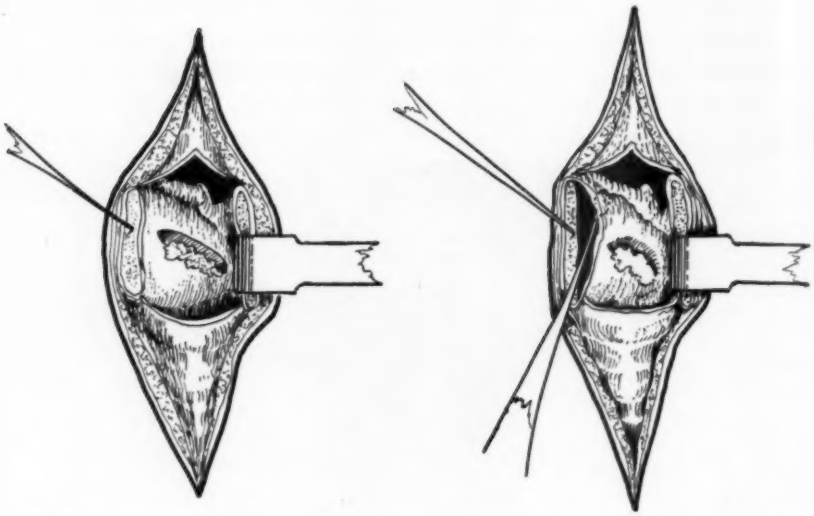


Fig. 3.—Schematic representation of laryngofissure by the clipping technic. Note the elevation of the internal perichondrium from the inner surface of the thyroid ala (Jackson and Jackson [4c]).

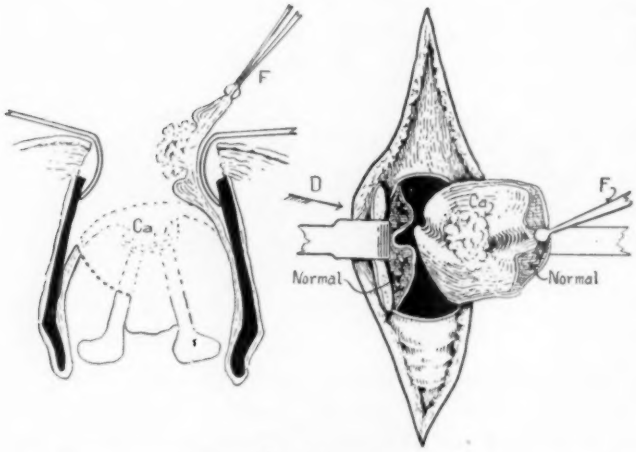


Fig. 4.—Schematic representation of laryngofissure by the anterior commissure technic. Note that soft tissues are cut first on the less involved side and that the flap is then reflected in such a way as to permit inspection of the lesion before the excision is completed (Jackson and Jackson [4c]).

examined before the excision is begun. In the clipping operation this is simple, but with the anterior commissure technic it is a little more difficult. In the former, one can inspect the lesion before doing even the subperichondrial dissection, but with the anterior commissure technic it is necessary first to separate off the internal perichondrium on both sides and then to section vertically *the less involved cord and ventricle,* so that the flap can be reflected in such a way as to permit good visualization of the entire interior of the larynx (fig. 4).

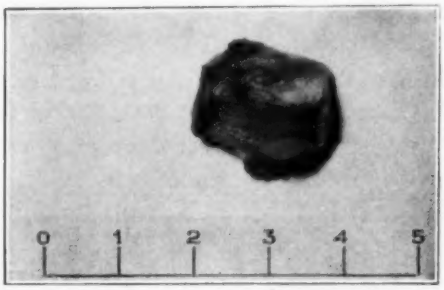


Fig. 5.—Operative specimen, showing vocal cord removed by the clipping technic. There was carcinoma of the middle third in an early stage (case 13).

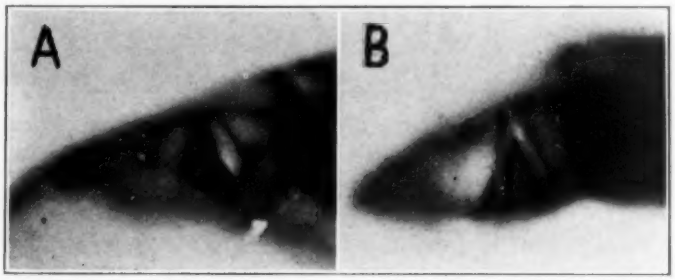


Fig. 6.—Photographs showing the postoperative appearance in 2 cases in which the right vocal cord was removed by laryngofissure (cases 20 [*A*] and 46 [*B*]).

This careful inspection permits one to decide on the extent of excision necessary to give a safe margin of normal tissue around the growth. In a number of cases I have removed only one cord and part of the ventricle, leaving the ventricular band untouched (figs. 5 and 6). I believe it permissible to do this in cases in which the growth is very limited.

Partial Removal of Cartilage.—No cartilage was resected in my series. Lack advocated resection of the thyroid ala along with the soft tissues bearing the growth, because he believed that it was easier to be

sure of adequate removal in this way. St. Clair Thomson has made it a part of his technic to do a subperichondrial resection of the thyroid ala before excising the tumor and adjacent soft tissues.

Excision of the Growth.—After subperichondrial dissection, the tumor-bearing mass is excised with special curved scissors, including generally the tip of the vocal process of the arytenoid on the more involved side. As already stated, in the case of the strictly cordal growth I feel that it is not necessary to include the ventricular band. If possible, the mass excised is kept in one piece, but of course it is necessary to remove additional tissue if it is found that the margin on one side or the other is insufficient.

Hemostasis.—Plain or iodoform gauze is tucked into the open larynx immediately after the excision, while the operative specimen is inspected. Generally, most of the bleeding will stop with this precaution alone. If a

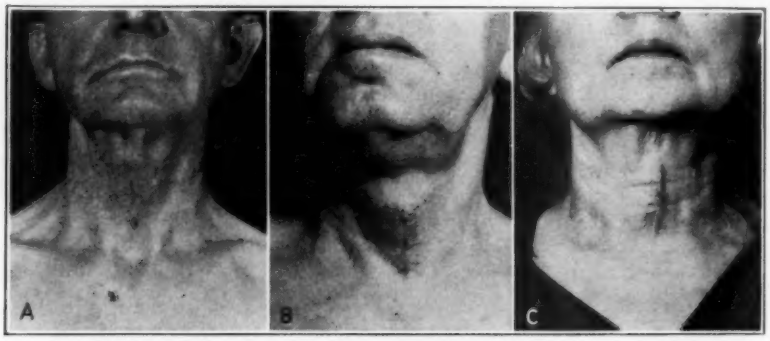


Fig. 7.—Postoperative appearance of neck at time of patients' discharge from the hospital (eighth to tenth day). The wound is closed without drainage and in most cases heals by first intention (cases 8 [*A*], 14 [*B*] and 16 [*C*]).

spurting vessel can be caught with a hemostat, so much the better, because later it may retract and be difficult to find. The larynx must be made absolutely dry before it is closed, or postoperative bleeding is sure to occur.

If the excision has been rather extensive, including one or both ventricular bands, the base of the epiglottis will be left without support and should be stitched forward, as suggested by Gabriel Tucker.[6a] This will tend to prevent anterior stenosis.

Closure.—No sutures are placed in the perichondrium or the cartilage. The fascial and muscular tissues are closed with buried sutures (alloy steel wire) and the skin with Michel clamps. Drainage

6a. Tucker, G.: Early Intrinsic Cancer of the Larynx: Diagnosis and Treatment; Observations on Laryngofissure as a Method of Treatment in a Series of Cases, Ann. Otol., Rhin. & Laryng. **41**:36-51 (March) 1932.

has not been provided for in the past several years (fig. 7), though in the early cases of the series a small wick of iodoform gauze was left in the lower end of the wound.

Chevalier Jackson [7] at one time favored packing the soft tissues down to the cartilage open, in order to allow free drainage for infection of the cartilage or the perichondrium and to lessen the formation of inward growing granulations.

Dressings.—A plain flat gauze dressing is applied, the first layer moistened with a 1 : 10,000 solution of mercury bichloride or compound tincture of benzoin. The dressings are changed daily, and the clips are removed on the third or fourth day.

St. Clair Thomson stated that he favored the omission of all dressing.

After-Care and Course.—The patient is kept sitting up, or rather the head of his bed is elevated. He is instructed not to talk but to use pad and pencil in expressing himself. After a few hours he is given liquids and soft solids, being encouraged to sit up, lean slightly forward and take a good swallow rather than to sip the liquid or take it through a glass tube. In none of my cases has a feeding tube been necessary, but if an extensive excision is made, especially if the arytenoid is removed, it may be necessary to use a feeding tube. The administration of opiates is contraindicated, and only the mildest sedation is permissible.

The wound is generally healed externally in four or five days (fig. 7), but the healing of the interior takes several weeks. Most patients are discharged from the hospital in from one week to ten days (see table).

COMPLICATIONS

Hemorrhage, if it occurs, generally occurs within the first twelve hours. It was observed in only 2 of the cases in this series. In these cases it was necessary to pack the larynx temporarily, but generally the bleeding point can be found and the vessel ligated. In these 2 cases packing seemed to have no harmful effects, though in 1 there was an especially active growth of granulations, requiring direct laryngoscopic removal. Obviously, it is necessary to insert a tracheal cannula if packing is done.

Granulomas form in about 35 per cent of the cases. Sometimes they shrink and disappear in a few weeks. They should be given a chance to do that, and if they do not they should be removed by direct laryngoscopy, for their presence, even though not obstructive to an important degree, will interfere with the development of voice.

Abscess, perichondritis and chondrial necrosis occur in a certain percentage of cases and require free drainage. Often the extrusion of a

7. Jackson, C.: (*a*) Peroral Endoscopy and Laryngeal Surgery, St. Louis, Laryngoscope Company, 1915; (*b*) footnote 6a.

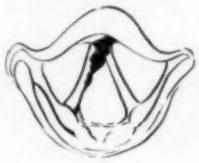
Data on Fifty Patients Operated on by Laryngofissure for Cancer of the Larynx

Case, Patient, Sex and Age (Yr.)	Location of Lesion	Type of Lesion	Operation Type, Date	Course	Result	Time Patient Has Remained Well
1 R. O. M 49		Squamous carcinoma	A (clipping) 8/20/30	10 days	Recurrence, 7/1/32; laryngectomy. Cure; well 8 yr. (1940) following laryngectomy or 10 yr. following original operation	10 yr.*
2 C. P. B. M 64		Squamous carcinoma grade 2	A 7/26/32	1 month; bronchopneumonia	Died 9/8/35 (heart); no recurrence	3 yr.†
3 W. S. M 53		Squamous carcinoma grade 2	A 6/21/34	12 days; granuloma removed twice	Cure	6 yr.
4 W. M. M 47		Squamous carcinoma grade 2	A 7/3/34	13 days; perichondritis 10 days later	Cure	6 yr.
5 H. Y. M 52		Carcinoma grade 2	A 8/9/34	15 days	Cure	6 yr.
6 H. W. F 52		Squamous carcinoma grade 4	A 9/28/34	14 days	Cure	6 yr.
7 J. G. M 46		Squamous carcinoma grade 2	A 10/30/34	13 days	Cure	6 yr.
8 W. V. M 54		Spindle cell carcinoma (fibroblastic)	A 12/4/34	9 days	Cure	5½ yr.

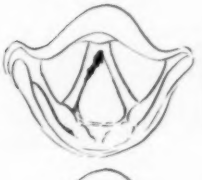
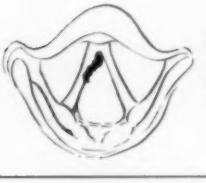
* Figure indicates the total period of survival since the original operation, but laryngectomy or second laryngofissure had been required for recurrence.

† Period of survival free of recurrence at time of death from other cause.

Data on Fifty Patients Operated on by Laryngofissure for Cancer of the Larynx—Continued

Case, Patient, Sex and Age (Yr.)	Location of Lesion	Type of Lesion	Operation Type, Date	Course	Result	Time Patient Has Remained Well
9 R. V. M 52		Squamous carcinoma (sarcomatoid)	B (Anterior commissure) 12/31/34	12 days	Recurrence 4/26/37; tracheotomy and laryngectomy; death postoperatively	2 yr.†
10 L. S. M 65		Squamous carcinoma grade 2	A 1/4/35	.....	Cure	5½ yr.
11 M. K. M 64		Squamous carcinoma grade 2	A 1/16/35	13 days	Death 1/38 (cardiac disease ?)	3 yr.
12 G. S. M 73		Squamous carcinoma grade 3	B 3/30/35	8 days; abscess incised by physician at home; granulomas removed; several direct laryngoscopic examinations	Cure	5½ yr.
13 H. McD. M 58		Squamous carcinoma grade 2	A 8/23/35	17 days; slight local infection	Cure	5 yr.
14 F. L. M 58		Squamous carcinoma grade 1	B 9/27/35	7 days	Cure	5 yr.
15 W. R. M 67		Leukoplakia; squamous carcinoma grade 2	A 11/21/35	6 days	Death 10/12/38	3 yr.†
16 C. H. F 63		Squamous carcinoma grade 2	A 4/17/36	8 days	Cure	4½ yr.

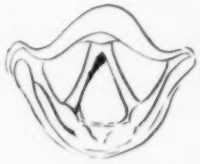
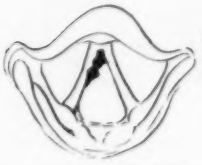
Data on Fifty Patients Operated on by Laryngofissure for Cancer of the Larynx—Continued

Case, Patient, Sex and Age (Yr.)	Location of Lesion	Type of Lesion	Operation Type, Date	Course	Result	Time Patient Has Remained Well
17 J. B. M 75		Squamous carcinoma grade 2	A 7/22/36	8 days; granuloma	Death 5/12/38; no recurrence	2 yr.†
18 A. B. F 38		Squamous carcinoma grade 3	B 10/2/36	10 days: small granuloma 1/8/37	Cure	4 yr.
19 F. O. M 58		Squamous carcinoma grade 2	A 10/22/36	10 days	Cure	4 yr.
20 M. B. F 49		Squamous carcinoma grade 1	A 2/20/37	10 days; granuloma	Cure	3½ yr.
21 L. P. F 44		Squamous carcinoma grade 1	A 3/6/37	14 days; hemostasis difficult; packing 48 hr.; granuloma removed several times	Cure	3½ yr.
22 W. N. M 47		Squamous carcinoma grade 1; leukoplakia laryngis	B 6/18/37	6 days; granuloma	Died 9/11/38 (cardiac)	1 yr.†
23 E. W. M 75		Squamous carcinoma grade 2	B 7/21/37	6 days; transient edema of the left arytenoid 9/19/37	Cure	3 yr.
24 E. Mc. M 57		Squamous carcinoma grade 2, showing leukoplakia	A 7/28/37	9 days	Cure	3 yr.

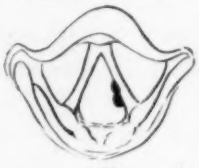
Data on Fifty Patients Operated on by Laryngofissure for Cancer of the Larynx—Continued

Case, Patient, Sex and Age (Yr.)	Location of Lesion	Type of Lesion	Operation Type, Date	Course	Result	Time Patient Has Remained Well
25 T. B. W. M 57		Squamous carcinoma grade 2	B 8/6/37	7 days; subcutaneous emphysema	Cure	3 yr.
26 H. H. M 43		Squamous carcinoma grade 3	B 8/11/37	14 days; slow drainage; granuloma 3 times	Cure	3 yr.
27 G. L. M 47		Squamous carcinoma grade 3	A 9/25/37	7 days	Cure	3 yr.
28 G. K. M 57		Squamous carcinoma grade 2	B 9/25/37	7 days; granuloma removed 12/13/37	Cure	3 yr.
29 R. L. M 39		Squamous carcinoma grade 1	A 10/19/37	6 days	Cure	3 yr.
30 A. P. M 56		Squamous carcinoma grade 2	A 10/27/37	13 days	Cure	3 yr.
31 W. R. B. M 60		Leuko-plakia; squamous carcinoma grade 1	B 11/29/37	10 days	Cure	2½ yr.
32 O. H. P. J. M 59		Squamous carcinoma grade 2	A 12/23/37	10 days	Died of aneurysm 1939; no recurrence	1½ yr.†

Data on Fifty Patients Operated on by Laryngofissure for Cancer of the Larynx—Continued

Case, Patient, Sex and Age (Yr.)	Location of Lesion	Type of Lesion	Operation Type, Date	Course	Result	Time Patient Has Remained Well
33 E. H. M 54		Squamous carcinoma grade 2	B 3/11/38	12 days	Cure	2½ yr.
34 P. L. J. M 63		Early carcinoma	A 4/16/38	10 days	Cure	2½ yr.
35 T. G. M 58		Squamous carcinoma grade 1	A 10/29/38	20 days; slow local infection; granuloma 12/29/38	Cure	2 yr.
36 W. J. M 53		Squamous carcinoma grade 1	B 11/5/38	14 days; hemorrhage; tracheotomy and packing; secondary closure	Cure	1½ yr.
37 S. L. M 69		Squamous carcinoma grade 4	B 1/31/39	17 days; fecal impaction	Well	1½ yr.
38 D. R. F 49		Squamous carcinoma grade 3	A 2/1/39	12 days; granuloma	Recurrence 8/26/40; second laryngo-fissure done	1½ yr.*
39 E. McC. F 47		Squamous carcinoma grade 2	B 2/28/39	8 days; granuloma removed several times; tracheotomy 4/11/39	Well	1½ yr.
40 G. B. M 69		Squamous carcinoma grade 2	A 3/14/39	9 days; very small granuloma	Well	1½ yr.
41 J. S. M 60		Squamous carcinoma grade 2	B 4/11/39	5 weeks; perichon-dritis and edema; granuloma	Well	1½ yr.

Data on Fifty Patients Operated on by Laryngofissure for Cancer of the Larynx—Continued

Case, Patient, Sex and Age (Yr.)	Location of Lesion	Type of Lesion	Operation Type, Date	Course	Result	Time Patient Has Remained Well
42 J. B. M 54		Squamous carcinoma grade 2	A 4/19/39	15 days; small granuloma	Well	1½ yr.
43 T. D. M 66		Squamous carcinoma	B 5/23/39	5 days	Recurrence 12/28/39; laryngectomy 1/2/40; well 9 mo. following laryngectomy, 1½ yr. following original operation	1½ yr.*
44 R. S. M 58		Squamous carcinoma grade 1	B 6/10/39	11 days	Well	1 yr.
45 S. E. M 45		Squamous carcinoma grade 2	B 6/14/39	16 days; granuloma	Well	1 yr.
46 J. M. M 65		Squamous carcinoma grade 2	A 8/26/39	8 days	Well	1 yr.
47 H. P. M 58		Squamous carcinoma grade 1	A 12/26/39	12 days	Well	9 mo.
48 R. G. M 71		Squamous carcinoma grade 2	A 2/27/40	8 days	Well	7 mo.
49 L. M. M 52		Squamous carcinoma grade 2	A 3/5/40	14 days; granuloma removed twice	Well	6 mo.
50 G. S. M 60		Squamous carcinoma grade 3	A 3/15/40	11 days	Well	6 mo.

little cartilaginous sequestrum will be followed by prompt healing, but in some cases a number of pieces come away before the wound will heal. I believe this complication should be treated conservatively.

Bronchopneumonia occurred in only 1 case of the series, though pulmonary complications (pneumonia, atelectasis and others) were formerly rather frequent and often fatal in patients who had undergone laryngofissure. The patient in my series recovered from his postoperative bronchopneumonia but died three years later of cardiorespiratory disease, with no recurrence of laryngeal cancer. The use of a local anesthetic and the withholding of postoperative opiates are probably factors in this improvement in the incidence of pulmonary complications.

RESULTS

In the series of 50 consecutive patients operated on by laryngofissure (table) there has been no operative mortality, but 6 patients have died of causes other than cancer, 3 of them having survived for three years, 1 for two years and 2 for a little over one year. In only 5 patients in the series have recurrences developed, and on 3 of these laryngectomy was subsequently done; 1 died of complications following the laryngectomy and the other 2 are still well, eight years and eight months after laryngectomy, or ten years and one and a half years, respectively, after the original operation. One has just had a second laryngofissure and may be given postoperative irradiation. Subtracting the last 4 patients, who were operated on less than a year ago and who are all free of any sign of recurrence but were too recently operated on to count, there remains a series of 46 patients, in only 4 of whom recurrences have developed. This gives 91 per cent well and free of recurrence for periods of one year and longer, not counting as cured the patients who have been well for eight years and one and a half years, respectively, after laryngectomy. Ten patients have survived, cancer free, for five years and over, 3 for four years, 13 for three years, 5 for two years and 11 for between one and two years. Since it is generally agreed that little significance can be attached to statistics recording cures of less than three years' duration, one may add the first three groups together. This gives 26 patients living and free of recurrence for at least three years, out of 30 patients treated by laryngofissure more than three years ago (prior to November 1937), an incidence of cures of 86.6 per cent.

Reviewing the literature from the point of view of results of laryngofissure, one finds that in the early days operative mortality was high, but even before 1900 Butlin and Semon had reduced it to practically nil. Recurrence was the rule in the early cases, both because of bad selection of patients for operation and because of technical factors. A great deal of attention was paid to the report by von Bruns (Berlin) which came out in 1878. Butlin and Semon, though at first of the same skeptical opinion

as many of their contemporaries, finally came to realize that the operation of laryngofissure has a definite place, and they set to work to make earlier diagnoses, to select their patients for operation more carefully and to perfect the technic. Finally, in 1903 Semon reported 18 cases (1891 to 1902), in 15, or 85 per cent, of which he had obtained a cure for one year or more. The next year Chevalier Jackson wrote: "Of all radical operations for the extirpation of intrinsic laryngeal growths this (thyrotomy) has given the best results, since its technic has been perfected and since its exact sphere of usefulness has been recognized." In 1909 Butlin reported 21 patients operated on between 1890 and 1908, with 1 operative death and 13 patients well after three years. In the same year Chiari reported 29 cases with 3 operative deaths and 11 recurrences; in 15 there was apparent cure. St. Clair Thomson reported his first series in 1912—10 cases without a mortality; 8 of the patients were well after one year and three months to seven and a half years. In 1914 Chevalier Jackson reported 27 cases with no operative deaths and 24 patients free of recurrence at the end of one year or longer. In one of their most recent publications, the Jacksons [4e] reported a series of 80 laryngofissures on suitable patients, with 1 operative death and freedom of recurrence for three years or longer in 80 per cent of the patients. Many other operators have reported series of cases in the past ten or fifteen years, and the percentage of cases in which cures were obtained varies from 75 to 90.

Voice.—Cure of the cancer is of course the primary consideration, but next comes the question of voice. All the patients in the series reported here were able to talk, but the quality of voice and its carrying power varied greatly. In some patients the postoperative voice was produced at the cordal level by a remaining good cord approximating with a cicatricial cord (fig. 6) or by two cicatricial cords. In other cases it was produced by approximation and vibration of the ventricular bands.

CONCLUSION

From my experience in a series of 50 cases, as well as from a study of the literature, I believe it is apparent that laryngofissure is a surgical procedure which gives cures in a high percentage of suitable cases of cancer of the larynx.

NASAL OBSTRUCTION AND IMPAIRMENT OF HEARING

REPORT OF FORTY-SIX CASES OF SUBMUCOUS RESECTION WITH AUDIOMETRIC STUDIES

MALCOLM R. JOHNSON, M.D.
RED WING, MINN.

"It is important to remember that pathology in the nose and nasal sinuses (the middle ear being one) is the cause underlying large numbers of deafened ears, be the deafness incipient, intermittent, permanent or progressive." [1] Literature contains many theories as to the causation of chronic types of hearing impairment. Many authors have stated definitely, or implied in their teachings, that nasal obstruction is a causative or at least a contributory factor in certain types of hearing deficiency. The relief of nasal obstruction by submucous resection or by other means is recommended by some otologists as one of the therapeutic measures in the treatment for chronic nonsuppurative otitis media.

The purpose of this paper is to review the literature pertaining to nasal obstruction with regard to the relation of this condition to impairment of hearing; to determine, by presenting a series of cases, whether there is a characteristic impairment of hearing caused by nasal obstruction, and to ascertain whether that deficiency in hearing can be relieved when the nasal obstruction is corrected by some such means as a submucous resection.

The word "deafness," as used in this paper, denotes impairment of hearing which incapacitates the person, and will not be used to refer to partial impairment of hearing, except as it appears in quotations.

It is apparent that the only way that nasal obstruction could interfere with hearing is by causing some involvement of the eustachian tube, with subsequent impairment of the ventilation of the middle ear. For a better understanding of the discussion from the literature reviewed on this subject, some of the anatomy of the eustachian tube and the physiology of the ventilation of the middle ear will be presented.

Submitted to the teaching faculty of the Graduate School of the University of Minnesota in partial fulfilment of the requirement for the degree of Master of Science in Otolaryngology, in June 1940.

1. Fowler, E. P.: Prevention and Management of Deafness, Tr. Am. Laryng., Rhin. & Otol. Soc. **38**:1, 1932.

The existence of the eustachian tube has been recognized since the sixteenth century, when Eustachius first described it; but it was not until 1920 that a clear picture of what really makes the tube function properly was recorded by Rich.[2]

> The tympanic membrane functions perfectly only when the atmospheric pressure is the same on both sides of it. . . . The equalization of pressure is made possible by the inflow of atmospheric air from the nasopharynx through the Eustachian tube into the tympanic cavity until the tension of the air within this cavity becomes identical with that of the air in the external auditory canal.
>
> . . . The orifices of the Eustachian tubes appear as slits in the mucous membranes of the nasopharynx. The two lips are plainly seen to lie normally in contact, i. e., the tubal orifice is normally closed when at rest.

After a series of careful experiments on animals and accurate clinical observations, Rich [2] came to the following conclusions:

1. Normally, the Eustachian tubes are closed. They are opened during swallowing, yawning, and sneezing reflexes. They exhibit no independent reflex dilatation either periodic or irregular. Normally, they are not opened by respiratory movements, either quiet or forced, and are unaffected by mouth breathing and simple elevation of the soft palate.

2. Although a most important function of the Eustachian tube is that of equalizing the atmospheric pressure on both sides of the tympanic membrane, the mere existence of a disturbed pressure equilibrium will bring about no regulatory dilatation of the tube, either independently or through the swallowing reflex. Deglutition, as a means of restoring the pressure equilibrium to the ear drum, is performed, when necessary, consciously or through habit; but this reflex is never set in motion directly by stimuli arising from the tension of the tympanic membrane.

3. The levator palati, the palatopharyngeus, the internal pterygoid, and the superior constrictor muscle of the pharynx were found to exert, by their contractions, *no* influence whatever upon the patency of the orifice or lumen of the tube.

4. The tensor palati is the only muscle which is functionally related to the Eustachian tube. Contraction of this muscle is always accompanied by dilatation of the tubal orifice and lumen. There exists no constrictor muscle of the tube. Relaxation of the tensor palati is followed by a passive return of the tube to the condition of approximation which they normally occupy when at rest.

Brown [3] stated that absorption of the content of air within the middle ear, even with constant renewal of the air content, is accepted as the most active factor in the development of retraction of the drum membrane.

Thompson, Howe, and Hughson [4] found that actual rupture of the drum membrane would occur if an intratympanic pressure of 60 to

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2. Rich, A. R.: A Physiological Study of the Eustachian Tube and Its Related Muscles, Bull. Johns Hopkins Hosp. **31**:206 (June) 1920.

3. Brown, F. E.: A Study of the Eustachian Tube with Special Reference to Recognition and Preservation of the Middle Ear, Northwest Med. **24**:25 (Jan.) 1925.

4. Thompson, E.; Howe, H. A., and Hughson, W.: Middle Ear Pressure and Auditory Acuity, Am. J. Physiol. **110**:312 (Dec.) 1934.

70 mm. of mercury was used. They maintained that there is no other closed cavity within the body which by absorption of air or by production of air pressure can produce changes in the pressure equivalent to 5 mm. of mercury above or below that usually present. They came to the following conclusions:

1. Either increased or decreased middle ear pressures, determined by direct measurement, impair the functional ability of the ear to approximately the same extent. This degree of impairment is directly proportional to the amount of pressure exerted.

2. Plus or minus 5 mm. of mercury was found to be the minimum pressure change which would produce impairment in the transmission of sound. Low and high frequencies are affected more than those in the middle range.

3. Variations in the middle ear pressure have no apparent effect on inner ear pressure nor is this effect modified by actually changing the inner ear pressure.

4. The effect of middle ear pressure change seems definitely directed against the conduction mechanism of the ear.

Armstrong and Heim,[5] in describing an occupational disease entity, defined aero-otitis media as an acute or chronic traumatic inflammation of the middle ear caused by a difference in pressure between the air in the tympanic cavity and that of the surrounding atmosphere, commonly occurring during changes in altitude in airplane flights. They said that the two principal causes of improper ventilation of the middle ear are (1) a failure to open the eustachian tube voluntarily when necessary and (2) inability to open it.

According to Armstrong and Heim,[5] inability to ventilate the middle ear voluntarily is much more prevalent than is generally recognized. Some of the most frequent causes of eustachian stenosis are acute and chronic infections of the upper respiratory tract, nasal obstructions, sinusitis, tonsillitis, tumors and growths of the nose and nasopharynx, paralysis of the soft palate or of the superior pharyngeal muscles, enlargement of the pharyngeal or of the tubal tonsil, inflammatory conditions of the eustachian tube or of the middle ear and scar tissue about the ostium of the eustachian tube following adenoidectomy.

In the Report of the Committee on the Problems of the Hard of Hearing from the Section on Laryngology, Otology and Rhinology of the American Medical Association,[6] one of the conclusions reached is this: "The most important etiologic factors according to the majority of otologists are nose and throat conditions that directly affect the Eustachian tube."

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5. Armstrong, H. G., and Heim, J. W.: Effect of Flight on the Middle Ear, J. A. M. A. **109**:417 (Aug. 7) 1937.

6. Hays, H.; Newhart, H.; Pierce, N.; Phillips, W., and Shambaugh, G.: Report of the Committee on the Problems of the Hard of Hearing, Section on Laryngology, Otology and Rhinology, J. A. M. A. **83**:2094 (Dec. 27) 1924.

Sluder [7] pointed out the close relationship of the wall of the sphenoid sinus to the eustachian tube and showed how infections of the sphenoid sinus could be an important causative factor in the production of conduction deafness.

To quote from Davis,[8]

> The prophylaxis of deafness or the conservation of hearing is only a method of applied preventive medicine in which the otologist is most actively engaged. The problem resolves itself into early recognition and correction of conditions which predispose to auditory disease. . . . In children the most potent cause of tubal inflammation is adenoids. So constant is this factor that the presence of congestion or retraction of the membrana tympani in children is regarded as one of the most reliable signs of lymphoid hypertrophy in the nasopharynx. If the adenoid mass is of sufficient size to interfere materially with nasal respiration, the drum membrane is usually retracted to a degree rarely seen in adults. With such findings, removal of the adenoid mass is definitely indicated.
>
> In older subjects, preventive methods must be directed toward the correction of those conditions that are conducive to repeated inflammations of the nasopharynx and to removal of foci of infection. Local applications to the nose and nasopharynx may be all that is necessary in a small number of cases, but where there are repeated attacks of Eustachian congestion, acute colds, and so on, one must consider seriously the removal of tonsils, correction of nasal deformities, and investigation of the sinuses and general physical condition.

Babbitt [9] concluded that the analysis of hearing impairment has reached a more satisfactory status than that with respect to treatment. "The sum total of probable relief seems greater in the conservation of hearing than in the treatment of established deafness."

According to Cary,[10] approximately 85 per cent of all deafness is due to middle ear involvement. Ninety per cent of these middle ear infections have their origin in inflammatory conditions of the nasopharynx with extension to the ear by way of the eustachian tube.

It has been suggested by Ivins [11] that a greater effort be made to prevent conditions which lead to inflammations of the eustachian tube and to prevent obstructions hindering drainage and ventilation. By such means, the tendency to inflammation of the eustachian tube and the nasal sinuses will be lessened, and mastoiditis and "dry catarrh" may be pre-

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7. Sluder, G.: Two Factors in Catarrhal Deafness, Tr. Sect. Laryng., Otol. & Rhin., A. M. A., 1926, p. 162.

8. Davis, J. E.: Different Types of Deafness: A Study of the Etiology, Methods of Diagnosis, Prophylaxis and Treatment, Laryngoscope **40**:20 (Jan.) 1920.

9. Babbitt, J. A.: Treatment of Non-Suppurative Deafness, Atlantic M. J. **28**:821 (Sept.) 1925.

10. Carey, E. H.: Some Otological Problems, Surg., Gynec. & Obst. **44**:770 (June) 1927.

11. Ivins, H. W.: Obstruction of the Nasal Passages with Special Reference to the Upper Regions, J. Iowa M. Soc. **13**:272 (July) 1923.

vented. Ivins expressed the belief that "dry catarrh" begins earlier in childhood than is ordinarily suspected—that nasal spurs and deflected and bulbous septums are notably in formation as early as from 4 to 5 years of age, becoming more marked as the child grows older.

In a paper on the prevention and treatment of chronic catarrhal otitis media, Barkhorn [12] stated,

> For the true catarrhal processes we must realize that sepsis is the underlying factor and that the main hope in preventing chronic middle ear deafness lies in the early recognition and treatment of catarrhal and septic changes in the upper respiratory tract.

He suggested that after tonsils and adenoids have been eliminated, roentgen examination should be made of the sinuses and doubtful ones explored. He was convinced that submucous resection is helpful only because of the improvement it brings about in the ventilation and drainage of the nasal sinuses, and not because of any change it may cause in the atmospheric pressure around orifices of the eustachian tubes.

Woods [13] said many patients came into his office complaining of deafness of variable degrees whose difficulty he attributed to closure of the eustachian tubes. His theory was that a deflection of the septum causes rarefaction of the air posterior to the point of obstruction which in time collapses the eustachian tube. When this closure becomes permanent, hearing begins to be affected. Furthermore, the discharge that usually accompanies a deflection of the septum may be forced into the eustachian tube with detrimental effects on hearing.

It is apparent from Woods's article that he was not familiar with Rich's work on the physiology of the eustachian tube. Woods spoke as if the eustachian tube were open at all times, being forced to close because of the rarefaction of the air behind a nasal obstruction. Actually the tube is opened by the muscular action of the tensor veli palatini, and an atmospheric pressure several times greater than normal is necessary to prevent the tube from being opened.

Shambaugh [14] stated that irregularities of the septum which have no clinical significance have often been held responsible for the development of progressive deafness in adults. His argument for this is simple:

> Since disease of the middle ear in children commonly results from diseased adenoids, it seems natural that one should look to the nose for the cause of defective hearing in adults, especially when a conduction type loss is present. This assumption is still more or less wide-spread, although it is based on two palpable errors. The condition known as progressive deafness in adults has, with few exceptions, nothing

12. Barkhorn, H. C.: Prevention and Treatment of So-Called Chronic Catarrhal Otitis Media, J. M. Soc. New Jersey **27**:118 (Feb.) 1930.

13. Woods, E. A.: The Causes and Results of Deflection of the Nasal Septum, Northwest Med. **20**:210 (Aug.) 1921.

14. Shambaugh, G. E.: Clinical Problems of Rhinology, Arch. Otolaryng. **16**:544 (Oct.) 1932.

to do with tubo-tympanic disease, but is the result, usually, of alterations that take place primarily in the bony capsule of the labyrinth. The second clinical error in this attempt to attribute the cause of progressive deafness to irregularities of the septum is the fact that obstructions to nasal respiration, per se, are no longer recognized as a possible etiologic factor in the production of tubo-tympanic alterations. . . . The clinical fact remains that no one has ever recorded an improvement in hearing from an operation on the nasal septum, and the plea for these operations is based on the rather flimsy proposition that operations for irregularities of the septum may retard the progress of the deafness. Such a proposition is not susceptible to proof or disproof. It is, however, not logical.

Love [15] expressed a similar opinion but favored doing the submucous resection, not to restore hearing in cases of chronic nonsuppurative otitis, but to benefit the nose itself and to improve the general health of the patient.

In a study of severe deafness (5,000 children in institutions for the deaf) Shambaugh [16] showed that the prevention of acquired deafness in childhood lies in the prevention and prompt treatment of acute infectious fevers, particularly during the first few years of life. The adults with profound deafness whom he observed were all members of the leagues of hard of hearing. Nearly all of those with conduction deafness showed secondary degeneration of the acoustic nerve. In a study of 165 patients in Washington, D. C., only 2 were found to have chronic sinusitis. He concluded that in only 12 per cent of adults with severe deafness was this condition due to disease of the middle ear beginning in childhood and in any way preventable by examination of school children. In 70 per cent of adults with severe deafness this handicap was due to otosclerosis.

In a discussion of the conservative treatment of chronic progressive deafness, Nash [17] reviewed his data on 200 patients who were under his personal observation for an average period of five years and ten months. In this careful report he gave a chart showing his very complete routine of treatments carried out on each patient. He stated that submucous resections did not improve the hearing in any of these patients. His conclusion was that the present conservative treatment of chronic progressive deafness is ineffective.

Walker,[18] on the other hand, expressed the belief that the primary cause of chronic progressive deafness is deficient and abnormal nasal

15. Love, J. K.: Discussion on Chronic Non-Suppurative Middle-Ear Deafness (Excluding Otosclerosis), Brit. M. J. **2**:1115 (Dec. 12) 1925.

16. Shambaugh, G. E.: Severe Deafness in Adults, Arch. Otolaryng. **18**:431 (Oct.) 1933.

17. Nash, C. S.: A Study in Treatment of Deafness, Tr. Am. Otol. Soc. **28**:107, 1938.

18. Walker, H.: Surgery of the Nasopharynx in the Treatment of Chronic Progressive Deafness, Laryngoscope **48**:552 (Aug.) 1938.

breathing. He stated that the problem, then, is to prevent abnormal breathing in the young and to correct the resulting deformities in the adult. He recommended electric coagulation of hypertrophied lower turbinates, submucous resection and even removal of small portions of the middle turbinates as means of halting the progress of deafness, because this procedure gives better drainage for the accessory sinuses and lessens nasopharyngeal congestion.

There is one fault to find with Walker's paper. His references include only those which support his point of view; none of these gave case reports to verify their statements. Walker's statements were apparently his clinical opinion and suspected results, but not his findings in actual cases.

In presenting 90 cases of marked obstruction of the eustachian tube, Ide [19] divided obstruction into two types—acute and chronic. There were 33 cases in which the obstruction was acute, and in only 1 was it associated with obstruction of the corresponding nares by a deviated septum. There were 57 cases in which the obstruction was chronic and in 14 there was a deviation of the nasal septum. In only 1 of these 57 cases was the presenting complaint nasal obstruction and stuffiness in the ears. Ide cited several cases, but none of those reported happened to be one in which nasal obstruction was shown.

The tendency to associate etiologically the occurrence of middle ear disease, including the more or less insidious form of middle ear "catarrh," with anatomic variations found in the nasal chambers is based on the theory that these variations cause middle ear disease by mechanically altering the air currents in the nasopharynx. Shambaugh [20] disagreed and presented as most convincing evidence against the air current theory 2 cases of congenital atresia of the choanae in which there was complete blockage of the air passage to the nasopharynx and no middle ear disease.

Richter,[21] in discussing the causes of difficulties in hearing, stated that a very common cause of typical sound conduction disturbance is occlusion of the pharyngeal end of the eustachian tube. This may be due to adenoid vegetations, tumor, hyperplasia of the posterior end of the turbinals, common inflammations, as colds, and other conditions. Local noninflammatory disease of the nose—deformities of the septum, for instance—cannot lead to occlusion of the tubes, hence cannot be a cause of a "hard of hearing" condition.

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19. Ide, C. E.: The Relation of Obstruction of the Eustachian Tube to Local and Systemic Conditions and to Prognosis Regarding Restoration of Hearing, Laryngoscope **27**:14 (Jan.) 1917.

20. Shambaugh, G. E.: The Relation of Nose and Throat to Ear Diseases, Illinois M. J. **42**:431 (Dec.) 1922.

21. Richter, H.: Causes of the Difficulties of Hearing, Med. Klin. **28**:885 (June 24) 1932.

In supporting the theory of focal infection, Emerson [22] tried to establish the idea that chronic deafness is essentially an infective process starting from a primary focus in the lymphoid tissue in which the barrier of the lymph nodes has been overcome; that the perception apparatus is damaged before deterioration of the conduction mechanism becomes a factor, and that chronic deafness is not steadily progressive but advances in cycles.

One of the most important steps in the prophylaxis of aural disease, according to Davis,[23] is the correction of those nasal abnormalities that interfere with the normal ventilation of the accessory sinuses.

Hoffmann [24] did not give deafness as an indication for submucous resection but in his discussion of contraindications stated:

> The submucous resection is contra-indicated as a general rule in patients who have a septal deflection and deafness in varying degrees which functional tests establish as perception deafness.

On the other hand, Hastings [25] expressed the opinion that removal of nasal obstruction or sepsis is likely to be of value in cases in which the hearing varies from day to day or in which immediate improvement in hearing follows the use of the eustachian catheter.

Six patients examined by watch tick were reported by Trowbridge [26] as having impaired hearing that improved after submucous resection. He expressed the belief that relief of nasal stenosis has brought about either improvement or cure in a great many cases of "deafness" which ordinarily would be considered hopeless.

Hitz [27] maintained that any nasal or postnasal obstruction to normal ventilation of the ears has its positive and negative phases. Nose blowing is a positive, or compressive, factor, while swallowing with an obstructed nose is a negative, or rarefying, factor. The correction of obstructive lesions in the nose, while helpful, will not be effective in controlling the downward trend of hearing, unless the patient consistently avoids pneumatic abuse.

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22. Emerson, F. P.: Is Chronic Progressive Deafness a Rhinologic or Otologic Factor? Ann. Otol., Rhin. & Laryng. **33**:865 (Sept.) 1924.

23. Davis, J. L.: Conservation of Hearing Made Practicable by the Timely Elimination of Nose and Throat Disorders, S. Clin. North America **4**:213 (Feb.) 1924.

24. Hoffmann, J. N.: Indications and Contra-Indications for Submucous Resection, Laryngoscope **33**:13 (Jan.) 1923.

25. Hastings, S.: The Preventive Medicine of the Ear, Brit. M. J. **1**:504 (March 14) 1925.

26. Trowbridge, D. H.: Effects of Septal Operations on Hearing, Ann. Otol., Rhin. & Laryng. **34**:513 (June) 1925.

27. Hitz, H. B.: Important Factors in the Conservation of Hearing, Wisconsin M. J. **38**:382 (May) 1939.

In the opinion of Shambaugh [28] the confusion of the more or less common tubotympanic processes, so common in childhood, with the chronic persisting processes which occur in adult life has been responsible for all sorts of "tinkering" operations on the nose for the relief of ear trouble, which his experience has shown to have nothing to do with nasal conditions.

Lapsley [29] stated that catarrhal forms of chronic ear trouble may result partly from adenoids, diseased tonsils, sinus trouble or various obstructions of the nose. "The results of nasal operations as an aid to relieving ear trouble seem very often disappointing."

Figure 1, taken from an article written by Crowe [30] in collaboration with Guild and Hughson, shows that increasing age is associated with impairment of hearing. Three hundred and fifty-three patients between

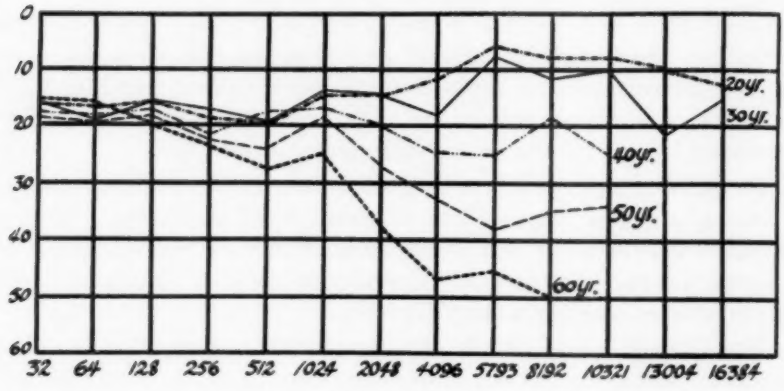

Chart 1.—The means for all age groups. Note that the low tones are heard equally well by all groups, while there is a drop in acuity for high tones in each successive age group. (After Bunch.)

the ages of 20 and 60 years with normal looking tympanic membranes and no history or subjective manifestations of aural disease were grouped according to each decade and the average acuity of hearing determined. The audiograms showed an increasing impairment of hearing for each successive age group. The results in other similar groups show that no one disease is responsible for causing the impairment of hearing for high tones that accompanies advancing age.

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28. Shambaugh, G. E.: Facts and Fancies in the Practice of Otolaryngology, J. A. M. A. **87**:1720 (Nov. 20) 1926.

29. Lapsley, R. M.: Progressive Deafness, J. Iowa M. Soc. **20**:160 (April) 1930.

30. Crowe, S. J.; Guild, S., and Hughson, W.: Investigations on the Underlying Causes of Deafness, in Harvey Lectures, 1931-1932, Baltimore, Williams & Wilkins Company, 1933, p. 100.

In a more recent article Crowe [31] emphasized the fact that in interpreting audiometer charts it is essential to know that with each decade the hearing for high frequencies becomes more and more impaired; that the three conditions in the middle ear that do impair hearing are partial or total obstruction of the eustachian tube, any lesion that interferes with the movement of the ossicles, and fixation of the footplate of the stapes due to otosclerosis.

In any discussion of the prevention of impairment of hearing, the recent work of Crowe and Baylor,[32] in which they used a special audiometer with a range of frequency from 32 to 16,384 double vibrations is outstanding—revolutionary. The classic accepted teaching in otology has always been that "impaired hearing for high tones with good hearing for low tones" always indicates a lesion of the inner ear or nerve. The repeated observation of this condition in children led Crowe and Baylor to doubt this theory. On further examination of these children they found an abnormal growth of lymphoid tissue partially obstructing the nasopharyngeal orifice of the eustachian tube. The earliest symptom of such partial obstruction was found to be impaired hearing for the frequencies between 10,000 and 16,000 double vibrations. Because the children had good hearing for all the lower and middle ranges of tone the condition remained undiagnosed. It was observed that if the partial obstruction was not treated, the loss in hearing would progressively involve one octave after another toward the low end of the scale, becoming evident only when the speech range was affected.

Crowe and Baylor recommended irradiation of these lymphoid hyperplasias by means of a radium bomb on a long metal applicator held in close proximity to the nasopharyngeal orifice of the eustachian tube under direct vision with the nasopharyngoscope. They reported remarkable results from this treatment and concluded that if the obstruction to the eustachian tube is recognized and properly treated before the age of 15, the hearing usually returns to somewhere near the normal level; after this age, however, the secondary changes in the middle ear may be so advanced that they cannot be repaired.

In speaking of nasal obstruction due to septal deflection, W. S. Syme [33] stated:

> I have for some time had an uneasy feeling that there is a tendency to blame this deformity for many more ills than it deserves. Middle ear and tubal conditions, especially catarrhal ones, are found with great frequency in association with septal deviation, but the suggestion is made that these are most commonly seen *after* the establishment of sinus disease.

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31. Crowe, S. J.: Diagnosis and Differential Diagnosis of Deafness, Arch. Otolaryng. **28**:663 (Nov.) 1938.

32. Crowe, S. J., and Baylor, J. W.: The Prevention of Deafness, J. A. M. A. **112**:585 (Feb. 18) 1939.

33. Syme, W. S.: Septal Deflection, Brit. M. J. **2**:656 (Sept. 24) 1938.

In my series of 46 cases of submucous resection, an attempt was made to find the relationship between nasal obstruction and impairment of hearing. The patients included in the study did not, on the whole, present themselves with aural complaints but came into the clinic complaining of inability to breathe properly through their noses. Complete histories and physical examinations were made in the medical clinic to rule out any complicating factors from systemic disease. In an effort to reduce the number of variable factors, the Wassermann test was required to be negative and the results of urinalysis, the hemoglobin content and the white blood cell count all had to be within normal limits before a patient was accepted for this series. Patients with a history of aural trouble (running ear, etc.) were excluded. Only those patients were accepted for this study who presented intact membrana tympani and nasal obstruction.

Routine roentgenograms of the sinuses were taken; audiograms were made in a partially sound-proofed room, lined with celotex, and overall hearing was tested with a Maico D-5 audiometer from 128 through 11,584 double vibrations. Bone conduction tests were not carried out because of the current controversy as to the interpretation of the findings. Weber and Rinne tests were done with a uniform stroke (impulse stimulus) on a 512 double vibration steel fork. The Rinne test was timed with a stopwatch so that a more accurate record could be made. In order to set up a control for this particular tuning fork, 20 normal persons were examined. It was found that air conduction averaged one hundred to one hundred and ten seconds and that bone conduction averaged thirty to forty seconds. This was taken as the normal positive observation with the Rinne test to be used in this series of cases. After the work-up was completed, a thorough submucous resection was done. No follow-up audiogram and recheck were done earlier than six months after the submucous resection. The average length of time for this recheck was eight and nine-tenths months.

Of the 46 patients, ranging in age from 14 to 54 years (average 30.3), 34 were male and 12 female. The duration of the nasal obstruction varied in the total number of patients from one year to thirty years, with an average period of ten and seven tenths years. All of the patients presented a common symptom, that of nasal obstruction or difficulty in breathing through the nose. This was accompanied by other presenting complaints, such as headache, postnasal discharge, sinusitis, frequent colds, frontal pain, intermittent sore throat, tinnitus, chronic cough, mouth breathing and snoring.

The audiograms were first checked for a characteristic impairment of hearing that could be considered the result of the nasal obstruction. None was found. To determine whether there was any improvement

in hearing for the group as a whole, averages were computed for the audiograms taken before submucous resection and for those taken at the recheck examination. The recheck audiogram was found to be, on

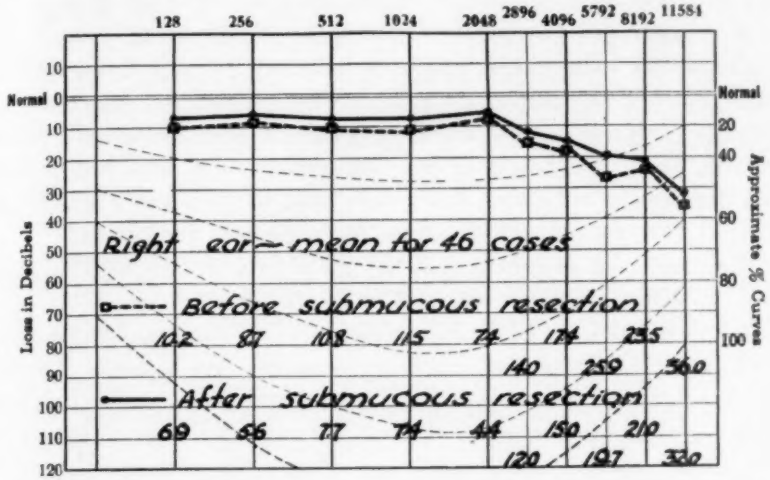
128 256 512 1024 2048 2896 4096 5792 8192 11581
Normal 0
Loss in Decibels
Approximate % Curves
Normal
Right ear — mean for 46 cases
Before submucous resection
102 87 108 115 74 174 235
140 259 360
After submucous resection
69 66 77 74 44 150 210
120 197 320

Chart 2.—Mean audiogram showing result before and after submucous resection (46 cases, right ear).

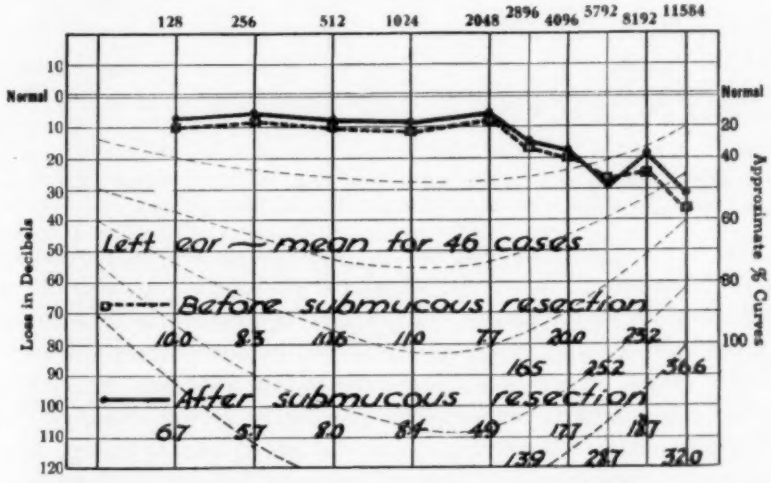
128 256 512 1024 2048 2896 4096 5792 8192 11584
Normal 0
Loss in Decibels
Approximate % Curves
Normal
Left ear — mean for 46 cases
Before submucous resection
100 83 116 110 77 200 252
165 252 366
After submucous resection
67 57 80 84 49 177 187
139 287 320

Chart 3.—Mean audiogram showing result before and after submucous resection (46 cases, left ear).

the average, 3 decibels higher then the original. This figure is well within the limits of experimental error. Often, too, a slight improvement is noted because of the fact that the patient is more familiar with the audiometer and the test procedure.

One of the most striking features of the recheck is the fact that 16 of these patients had perfectly normal audiograms that did not change. Fourteen others showed normal audiograms except for a high frequency tonal dip. Six of these showed no change in the recheck audiograms, and 8 showed slight (5-10 double vibrations) improvement. There were 7 patients who showed a definite loss in acuity for high tones with no improvement in the audiogram following the submucous resection. In 3 of these cases a diagnosis of definite traumatic deafness due to repeated high frequency trauma was made. There were 6 patients who showed some impairment of hearing with slight improvement and 3 with slight impairment of hearing and no change in the recheck audiogram.

The submucous resections gave definite positive results with improvement of the patient's general condition and improvement of his nasal physiology by allowing better breathing space. A number of the patients stated that their colds and attacks of sinusitis were lessened. A few were benefited by relief from the postnasal drip and chronic cough of which they had complained. Of the 3 patients who complained of tinnitus, 1 was relieved. With proper indications, the submucous resection is regarded by many as the most beneficial of nasal operations.

Chronic progressive infection of the nasal and pharyngeal mucous membranes was termed by Mosher [34] an "unsolved problem." Concerning it, he wrote,

> He who can arrest or cure this condition will prevent or cure chronic progressive deafness. Such an achievement would be a monument to any man. Those who have been in practice fifteen years or more can easily recall cases of progressive infection of the nasal and pharyngeal mucous membrane for which your minutest search gave no cause, that is, no focus of infection, and for which no remedy was of avail. . . . There was slow but steady loss of hearing.

The removal of a nasal obstruction in a case of chronic nonsuppurative otitis media can in no way affect the pathologic condition already present within the middle ear. Pathologic processes can be diagnosed as progressive or stationary only after continuous observation over a period of years. In only 1 of the 46 cases in this study was the condition chronic nonsuppurative otitis media. During the period of one year following the submucous resection there was no significant change in the audiogram. Presenting only 1 case of this type, it is not within the scope of this paper to offer any suppositions or prognostications as to the future progression of the impairment of hearing in chronic nonsuppurative otitis media.

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34. Mosher, H. P.: Unsolved Problems in Otolaryngology, Tr. Am. Acad. Ophth. **42**:27, 1937.

SUMMARY

The literature pertaining to nasal obstruction and its relation to impairment of hearing has been reviewed. It is apparent that nasal obstruction could produce impairment of hearing only by causing some disturbance in the nasopharyngeal orifice of the eustachian tube. Most authors agree that the presence of an anatomic obstruction in the nose cannot affect the eustachian tube either by misdirecting the air currents in the nasopharynx or by causing inflammatory reactions in the tube. If, on the other hand, this obstruction causes a prolongation of infections of the upper respiratory tract, so that they more frequently go on to sinusitis, it is likely that the presence of infections in the nose and in the nasopharynx would cause inflammatory reactions in the tube that might lead to adhesive processes in the middle ear. The inflammatory change, therefore, has been the result of the infection and not the direct result of the nasal obstruction.

A review of 46 cases of submucous resection for nasal obstruction is presented. In 65 per cent (30 cases) there was either no deficiency in hearing or only high tonal dips. In the cases in which some impairment of hearing was present the audiometric improvement was not sufficient to warrant urging the operation as a means of improving hearing.

CONCLUSIONS

1. Nasal obstruction per se does not cause a characteristic impairment of hearing.

2. Submucous resection is not to be recommended as a means of improving deficiencies in hearing.

Dr. Horace Newhart, head of the Department of Otolaryngology, University of Minnesota, and Dr. Malcolm C. Pfunder, chief of ophthalmology and otolaryngology at Minneapolis General Hospital, gave encouragement and valuable advice. Dr. H. I. Lillie, of Rochester, Minn., gave constructive criticism.

Interstate Clinic.

ROENTGEN RAY THERAPY FOR PATIENTS WITH CHRONIC SINUSITIS

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Roentgen radiation has been used in the treatment of a great many conditions. Through clinical observation and animal experimentation the action of various amounts on different types of tissue has been determined. The action of roentgen rays, even in small amounts, is always primarily destructive.[1]

The higher the specialization of the cell the less developed is its power of reproduction; therefore, the less sensitive is it to irradiation. The order of the degree of sensitivity of cells is as follows:[2]

(1) lymphoid cells (lymphocytes), (2) polymorphonuclear and eosinophilic leukocytes, (3) epithelial cells, (4) endothelial cells of blood vessels, (5) connective tissue cells, (6) muscle cells, (7) bone cells and (8) nerve cells.

Heine[3] has shown that the cilia of the respiratory epithelium are not destroyed by large doses of roentgen rays.

Pohle and Ritchie[4] have shown experimentally that a dose of 1,000 roentgens (r) at one sitting one to thirty days before incision did not influence healing perceptibly but that the same dose twenty-four to forty-eight hours after incision retarded healing, though it did not change the end result. They also found marked variation in the responses of animals receiving like dosage.

Desjardins[5] stated the belief that inflammatory lesions respond to roentgen ray therapy in proportion to the degree of leukocytic infiltration. In his experience furuncle, carbuncle, phlegmonous cellulitis, acute epi-

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From the Grand Forks Clinic.

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1. Dietz, T. J.: Volume Increase of Bacteria from X-Ray Irradiation, Radiology **24**:31 (Jan.) 1935.

2. Kaplan, I. L.: Radiation Therapy, New York, Oxford University Press, 1937, p. 20.

3. Heine, L. H.: The Effect of Radiation upon Ciliated Epithelium, Ann. Otol., Rhin. & Laryng. **45**:60 (March) 1936.

4. Pohle, E. A., and Ritchie, G.: Studies of the Effect of Roentgen Ray on the Healing of Wounds, Radiology **20**:102 (Feb.) 1933.

5. Desjardins, A. N.: The Action of Roentgen Ray or Radium on Inflammatory Processes, Radiology **29**:436 (Oct.) 1937.

demic infectious adenitis, onychia, paronychia and infection from gas bacilli are the acute inflammatory lesions which are favorably influenced. Tuberculous adenitis, tuberculous keratitis, tuberculous iritis, actinomycosis and blastomycosis are some of the chronic conditions favorably influenced.

Fenton and Larsell,[6] in collaboration with Butler and Wooley, subjected a series of cats with unilateral frontal sinusitis to roentgen ray therapy and found that marked destruction of lymphocytes and a considerable influx of large polyblasts and histiocytes occurred. They stated that they felt that the massive mobilization of histiocytic elements in the early stages is for the purpose of phagocytosing destroyed lymphocytes; the histiocytes are possibly attracted by the disintegration products.

Soto[7] and his associates, experimentally studying the effects of roentgen ray therapy on acute pyogenic infections of the skin and subcutaneous tissues, came to the conclusion that benefits from this form of therapy were due not to destruction of the lymphocytes and liberation of antibodies which they contained but to reduction of edema and products of inflammation through action on the capillary bed.

Butler and Wooley,[8] reporting on 450 of 700 patients with chronic sinusitis treated with roentgen ray therapy, observed that 36 per cent were cured, 55 per cent improved and 9 per cent slightly improved or not helped. They stated the belief that patients with hyperplastic membranes are the most likely to be helped, while those with polyps, atrophic rhinitis and chronic sinusitis with a fibrotic condition of the membranes and those who have been unsuccessfully treated by radical surgical procedure are least likely to be helped.

Smith and Nickel[9] treated 25 patients for sinusitis and reported good results in every case. They came to the conclusion that the most satisfactory results were secured in those in whom the condition resembled acute vasomotor rhinitis.

Hodges[10] divided his cases, for therapeutic purposes, into four groups. Group 1 is made up of cases of acute sinusitis and sinusitis in

6. Fenton, R. A., and Larsell, O.: The Histopathological and Clinical Interpretation of Experimental Sinus Disease, Tr. Am. Acad. Ophth. **38**:329, 1933.

7. Soto, J. A.; Brunschwig, A., and Schlutz, F. W.: Experimental Study of Effects of X-Radiation on Acute Pyogenic Infection of Skin and Subcutaneous Tissues, Surgery **3**:593 (April) 1938.

8. Butler, F. E., and Wooley, T. M.: Roentgen Therapy in Chronic Sinusitis, Radiology **23**:529 (Nov.) 1934; The Roentgen Treatment of Chronic Sinusitis, ibid. **30**:686 (June) 1938.

9. Smith, H. B., and Nickel, A. C.: Treatment of Subacute and Chronic Sinusitis by Roentgen Radiation, Am. J. Roentgenol. **39**:271 (Feb.) 1938.

10. Hodges, F. M.: Roentgen Therapy of Infections of the Nasal Accessory Sinuses, Am. J. Roentgenol. **39**:578 (April) 1938.

children, and he remarked that small doses of roentgen rays shorten the period of convalescence from acute sinusitis. His results in children who were not allergic were uniformly good. In group 2 he placed cases of secondary bronchitis or early bronchiectasis, and he concluded that the results obtained warrant the use of roentgen ray therapy before the patient is submitted to surgical treatment. In group 3 he placed cases of atrophic rhinitis. He seemed to feel that in the treatment of this condition roentgen radiation is of no value. Group 4 is made up of cases of hyperplastic sinusitis with early polypoid changes. The author's results in this group were only fair.

Rathbone [11] treated 70 children for sinusitis and reported 57 per cent cured, 28 per cent improved, 15 per cent not benefited; McLendon [12] treated 50 children and reported 27 to 44 per cent cured, 20 to 40 per cent much improved, 5 to 10 per cent slightly improved; Marks,[13] Warren,[14] Koch,[15] all reported varying degrees of success with this form of therapy.

Very little of the reported work on this subject is to be found in the otolaryngologic literature, and one wonders if many patients were not treated on the basis of roentgen diagnosis alone.

Kornblum [16] said:

> Most radiologists will readily admit that in the entire field of roentgenology nothing affords them greater difficulty than examination of the paranasal sinuses. . . . I was somewhat concerned with the difficulty I was having in the interpretation of sinus films and in order to check my own interpretations I made it a practice to show sinus roentgenograms first to Dr. E. P. Pendergrass and then to Dr. H. K. Pancoast my mentors in radiology, getting their opinions independent of each other and was surprised to find how seldom the three of us were in complete agreement.

All the 75 patients in my series were studied clinically and in most instances were followed for some time before being referred for roentgen ray therapy. Every known means, including roentgenograms when indicated, was used to arrive at a correct diagnosis, and only those patients whose diagnosis was beyond doubt were submitted to roentgen

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11. Rathbone, R. R.: Roentgen Therapy of Chronic Sinusitis in Children, Am. J. Roentgenol. **38**:102 (July) 1937.

12. McLendon, P. A.: Chronic Sinusitis in Children with Special Reference to X-Ray Therapy, M. Ann. District of Columbia **7**:341 (Nov.) 1938.

13. Marks, H. B.: Chronic Sinusitis in Children, New England J. Med. **216**: 604 (April 8) 1937.

14. Warren, E. D.: Value of X-Ray Therapy in Chronic Sinusitis, Laryngoscope **45**:864 (Nov.) 1935.

15. Koch, J.: Klinische und experimentelle Erfahrungen über die Roentgenbestrahlung akuter und chronischer Nebenhöhlenerkrankungen, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **143**:385, 1937.

16. Kornblum, K.: A Roentgenologist Looks at Sinus Disease, Am. J. Roentgenol. **38**:48 (July) 1937.

ray therapy. Many of those treated were definitely known to be allergic, although no extensive studies of allergy were made.

At first my impression of roentgen therapy was uniformly poor. I later found that I had formed an opinion too soon after the treatment. At least two to three months should elapse before one definitely concludes that the therapy has failed, although beneficial results may be noted before this.

Most patients in my series were treated with the following technical factors: 150 kilovolts, 20 milliamperes, 50 cm. distance, 0.25 mm. of copper plus 1 mm. of aluminum filter, half value layer of 0.46 mm. copper, 31 r per minute. For some patients with polyps or with a pathologic condition of the posterior sinuses, higher voltage was used in some treatments, the factors then being 200 kilovolts, 20 milliamperes, 50 cm. distance, 0.5 mm. of copper plus 1 mm. aluminum filter, half value layer of 0.95 mm. copper, 44 r per minute. All roentgen doses are given as measured in air. All children were treated with the low voltage. Adults and older children when treated were in the anteroposterior position, with a lead mask to protect the nose, eyes and eyebrows and hair lines. Younger children were treated with a 10 by 10 cm. cone, the eyes and eyebrows only being protected. Some of the children were treated from the sides as well as from the front.

In the treatment of adults the doses given varied somewhat with the stage of infection, the temperament of the patient and the distance the patient had to come for treatment. Patients with a history suggesting recent acute infection were given smaller doses, repeated at seven to ten day intervals for a number of treatments. Those with long-standing disease and those who lived long distances away were usually treated with a single heavy dose, repeated in one to three months if necessary.

All children were treated with smaller, repeated doses. I am not yet able to give an opinion as to which technic produces the best results.

The dates of treatment and factors used for each patient are given in the tables.

No harmful effects were noted in any of my patients, other than mild headache, which developed in a small number. This condition cleared up rapidly except in 2 patients, in whom it lasted nearly a week and was accompanied by slight nausea. Most of those treated had a marked increase in nasal secretion, lasting from the second to the seventh day. In patient 8 the discharge became so thick and was produced in such large quantities that it had to be removed by suction to relieve the patient's nasal obstruction.

Some interesting observations were made in the case of patient 16. Before treatment she complained of frontal headaches, postnasal dis-

TABLE 1.—*Summary of Data on Patients with Chronic Sinusitis Who Were Treated with Roentgen Rays*

No.; Age, Yr.; Sex	Duration of Symptoms	Results of Treatment	Time Followed After Treatment	Dates of Treatment	Dose, (r)	Kv.	Filter, Mm.	Half Value Layer of Copper, Mm.	Per R Min.	Ma.
1 7 M	2 yr.	Cure	1½ yr.	11/27/37 12/ 1/37 12/ 8/37	157 157 157	150	0.25 Cu, 1 Al.	0.46	31	20
2 16 M	3 yr.	Cure	1 yr.	2/ 3/38 2/ 8/38 2/14/38	200 200 200	150	0.25 Cu, 1 Al.	0.46	31	20
3 14 F	4 mo.	Failure	1 yr.	2/16/38 3/28/38 3/14/38	300 300 250	150	0.25 Cu, 1 Al.	0.46	31	20
4 4 M	2 yr.	Failure	2 mo.	10/ 2/37 10/ 7/37 10/12/37	150 150 200	150	0.25 Cu. 1 Al.	0.46	31	20
5 16 M	3 mo.	Marked improvement	5 mo.	3/ 5/38 3/14/38 3/21/38 8/22/38	250 250 250 300	150	0.25 Cu, 1 Al.	0.46	31	20
6 5 M	2 yr.	Cure	8 mo.	4/22/38 4/29/38 5/ 6/38	200 200 250	150	0.25 Cu, 1 Al.	0.46	31	20
7 10 M	2 yr.	Cure	10 mo.	2/ 4/38 2/ 9/38 2/14/38	250 250 350	150	0.25 Cu, 1 Al.	0.46	31	20
8 15 F	6 to 8 mo.	Cure	6 wk.	12/20/38 12/27/38 1/ 3/39	250 200 200	150 150 200	0.25 Cu, 1 Al. 1 Al.	0.46 0.46 0.95	31 31 21	20 20 10
9 9 M	1 yr.	Marked improvement	6 wk.	8/16/38	300	150	1 Al.	0.46	31	20
10 8 M	Years	Cure	1 yr.	12/28/37 1/ 3/38 1/ 6/38	200 200 200	150	0.25 Cu, 1 Al.	0.46	31	20
11 14 M	Years	Marked improvement	2 mo.	11/14/38 11/31/38 12/ 2/38	200 200 250	150	0.25 Cu, 1 Al.	0.46	31	20
12 6 M	Years	Failure	5 mo.	8/26/38 8/31/38 9/ 6/38	200 200 200	150	0.25 Cu, 1 Al.	0.46	31	20
13 13 M	1 yr.	Cure	8 mo.	2/17/38 2/22/38 3/ 1/38	250 250 250	150	0.25 Cu, 1 Al.	0.46	31	20
14 10 M	3 mo.	Cure	6 mo.	7/ 8/38 7/14/38	200 300	150	0.25 Cu, 1 Al.	0.46	31	20
15 14 M	2 to 3 yr.	Cure	1 yr.	2/ 1/38 2/ 5/38	300 300	150	0.25 Cu, 1 Al.	0.46	31	20
16 20 F	4 yr.	Marked improvement	1 yr.	3/ 4/37	630	200	0.25 Cu, 1 Al.	0.95	44	20
				1/10/38	450	150	0.25 Cu, 1 Al.	0.46	31	20
				5/ 3/38	400	150	0.25 Cu, 1 Al.	0.46	31	20
17 23 F	2 yr.	Failure	6 mo.	2/ 7/38 5/16/38	450 450	150	0.25 Cu, 1 Al.	0.46	31	20
18 18 F	6 mo.	Failure	6 mo.	4/18/38 4/22/38 4/29/38 5/21/38 5/30/38	200 250 200 158 150	150	0.25 Cu, 1 Al.	0.46	31	20
19 23 F	Years	Marked improvement	4 mo.	2/18/38	450	150	0.25 Cu, 1 Al.	0.46	31	20
				3/24/38	450	150	0.25 Cu, 1 Al.	0.46	31	20
				7/ 5/38	450	200	0.25 Cu. 1 Al.	0.95	44	20

TABLE 1.—*Summary of Data on Patients with Chronic Sinusitis Who Were Treated with Roentgen Rays—Continued*

No.; Age, Yr.; Sex	Duration of Symptoms	Results of Treatment	Time Followed After Treatment	Dates of Treatment	Dose, (r)	Kv.	Filter, Mm.	Half Value Layer of Copper, Mm.	Per R Min.	Ma.
20 40 F	Years	Marked improvement	1 yr. 2 mo.	12/18/38	450	150	0.25 Cu, 1 Al.	0.46	31	20
21 22 F	Years (allergic?)	Marked improvement	5 mo.	5/31/38	300	150	0.25 Cu, 1 Al.	0.46	31	20
22 35 F	Years	Marked improvement	1 mo.	4/20/38	440	200	0.5 Cu, 1 Al.	0.95	22	10
23 30 F	Years	Marked improvement	1 yr.	8/28/37	460	150	0.25 Cu, 1 Al.	0.46	31	20
24 33 F	Years	Marked improvement (allergic)	8 mo.	4/22/38	400	150	0.25 Cu, 1 Al.	0.46	31	20
25 36 F	Years	No improvement (allergic)	Patient antagonistic; unable to observe	4/27/38 5/ 2/38 5/11/38	350 200 200	150	0.25 Cu, 1 Al.	0.46	31	20
26 23 F	2 yr.	Marked improvement	5 mo.	4/14/37	450	150	0.25 Cu, 1 Al.	0.46	31	20
27 40 M	Years	No improvement	3 mo.	11/14/38	400	150	0.25 Cu, 1 Al.	0.46	31	20
28 28 M	Years	Cure	6 mo.	7/15/38 8/12/38	350 250	150	0.25 Cu, 1 Al.	0.46	31	20
29 20 M	6 mo.	Failure	4 mo.	9/29/37 11/15/37	500 500	150	0.25 Cu, 1 Al.	0.46	31	20
30 39 F	3 mo.	Cure	6 mo.	3/29/37 5/22/37	500 400	150	0.25 Cu, 1 Al.	0.46	31	20
31 22 M	Years	Marked improvement	4 mo.	1/12/38 6/24/38	474 374	150 200	0.5 Cu, 1 Al.	0.46 0.95	31 22	20 10
32 36 F	Years	Marked improvement	2 mo.	1/ 3/39 1/10/39 1/17/39	200 200 200	150	0.25 Cu, 1 Al.	0.46	31	20
33 28 M	1 yr.	Failure	4 mo.	5/26/38 6/ 2/38	300 300	150	0.25 Cu, 1 Al.	0.46	31	20
34 32 M	Years	Failure	1 yr.	5/12/37 8/28/38	400 440	150 200	0.25 Cu, 1 Al. 0.25 Cu, 1 Al.	0.46 0.95	31 44	20 20
35 38 M	Years	Failure	6 mo.	7/ 8/38	400	150	0.25 Cu, 1 Al.	0.46	31	20
36 23 F	Years	Failure	1 yr.	2/ 7/38	450	150	0.25 Cu, 1 Al.	0.46	31	20
37 28 M	Years	Marked improvement without symptoms for 6 mo.	1 yr.	11/13/36 2/12/37	330 550 500	140 200	4 Cu, 0.42 Al. 0.5 Cu, 1 Al.	.... 	.. 	..
38 29 F	3 yr.	Marked improvement	1 yr. 6 mo.	8/12/37	450	150	0.5 Cu, 1 Al.	0.46	41	20
39 23 F	1 yr.	Failure	1 yr.	12/22/37 9/ 8/38	450 400	150	0.5 Cu, 1 Al.	0.46	41	46

TABLE 1.—*Summary of Data on Patients with Chronic Sinusitis Who Were Treated with Roentgen Rays—Continued*

No.; Age, Yr.; Sex	Duration of Symptoms	Results of Treatment	Time Followed After Treatment	Dates of Treatment	Dose, (r)	Kv.	Filter, Mm.	Half Value Layer of Copper, Mm.	Per R Min.	Ma.
40 37 M	Years	Marked improvement	10 mo.	2/19/38 4/30/38 8/28/38	500 500 350	200 150	0.5 Cu, 1 Al. 0.25 Cu, 1 Al.	0.95 0.46	44 41	20 20
41 37 M	Years	Failure	6 mo.	3/ 1/38 4/ 7/38	450 400	150 200	0.25 Cu, 1 Al. 0.5 Cu, 1 Al.	0.46 0.95	31 44	20 20
42 39 F	1 yr.	Marked improvement	4 mo.	8/24/38 9/ 1/38	300 300	150	0.25 Cu, 1 Al.	0.46	31	20
43 21 F	6 mo.	Cure	1 yr.	6/ 1/37	450	150	0.25 Cu, 1 Al.	0.46	31	20
44 36 M	Years	Marked improvement	1 yr.	2/16/38	500	200	0.5 Cu, 1 Al.	0.95	44	20
45 38 M	3 mo.	Failure	2 mo.	4/12/38	300	150	0.25 Cu, 1 Al.	0.46	31	20
46 40 F	Years	Failure	4 mo.	7/26/38 8/10/38	250 400	150 200	0.25 Cu, 1 Al. 0.5 Cu, 1 Al.	0.46 0.95	31 44	20 20
47 36 F	6 mo.	Failure	3 mo.	8/17/38	400	150	0.25 Cu, 1 Al.	0.46	31	20
48 29 M	4 yr.	Failure	6 wk.	1/11/39	400	150	.....	....	..	..
49 39 F	6 mo.	Marked improvement	4 mo.	7/14/38 7/25/38 8/26/38	... 	150 200 200	0.25 Cu, 1 Al. 1 Al. 1 Al.	0.46 0.95 0.95	31 44 44	20 20 20
50 33 F	Years	Cure	9 mo.	3/19/37	450	150	1 Al.	0.46	31	20
51 36 F	1 yr.	Cure	2 yr.	3/19/37	500	200	1 Al.	0.95	44	20
52 37 M	Years	Marked improvement	5 mo.	4/22/38 5/26/38 7/27/38	450 250 300	150	1 Al. 1 Al. 0.5 Cu.	0.46 0.95	31 44	20 20
53 30 F	1 yr.	Marked improvement	6 wk.	6/30/38 7/ 5/38 7/12/38	200 200 200	150 200 150	0.25 Cu 0.5 Cu 0.25 Cu	0.46 0.95 0.46	31 22 31	20 10 20
54 34 F	Years	Marked improvement	1 yr. 6 mo.	1/18/37 1/21/37	300 300	200	0.5 Cu, 1 Al.	0.95	34	20
55 33 M	14 yr.	Marked improvement	6 mo.	7/ 7/38 8/ 6/38 8/15/38	150 200 300	150	0.25 Cu, 1 Al.	0.46	31	20
56 36 M	10 yr.	Marked improvement	5 mo.	9/22/38	450	150	0.25 Cu, 1 Al.	0.46	31	20
57 22 M	6 mo.	Marked improvement	11 mo.	3/14/38	450	150	0.25 Cu, 1 Al.	0.46	31	20
58 48 F	Years	Marked improvement	6 mo.	5/31/38 8/14/38	200 158	150	0.25 Cu, 1 Al.	0.46	31	20
59 57 M	1 yr.	Cure	6 mo.	10/ 1/37 11/30/37 5/10/38	500 475 500	150 150 200	0.25 Cu, 1 Al. 0.25 Cu, 1 Al. 0.5 Cu	0.46 0.46 0.95	31 31 44	20 20 20

TABLE 1.—*Summary of Data on Patients with Chronic Sinusitis Who Were Treated with Roentgen Rays—Continued*

No.; Age, Yr.; Sex	Duration of Symptoms	Results of Treatment	Time Followed After Treatment	Dates of Treatment	Dose, (r)	Kv.	Filter, Mm.	Half Value Layer of Copper, Mm.	Per R Min.	Ma.
60 49 F	16 yr.	Marked improvement	2 mo.	6/23/38	250	150	0.25 Cu, 1 Al.	0.46	31	20
				6/30/38	200	150	0.5 Cu,	0.46	31	20
				7/ 7/38	200	200	1 Al.	0.95	22	10
				8/19/38	300	150	0.25 Cu	0.46	31	20
61 42 F	6 mo.	Marked improvement	8 mo.	4/23/38	450	150	0.25 Cu	0.46	31	20
62 59 F	Years	Marked improvement	8 mo.	7/27/38	400	150	0.25 Cu	0.46	31	20
				11/ 3/38	400					
63 49 F	Years	Marked improvement	6 mo.	3/23/38	250	150	0.25 Cu	0.46	31	20
				4/ 7/38	350	200	0.5 Cu	0.95	44	20
				4/20/38	350	200	0.5 Cu	0.95	44	20
64 60 F	Years	Failure	7 mo.	3/ 5/38	352	200	0.5 Cu	0.95	44	20
				3/15/38	308					
				4/ 2/38	308					
65 50 M	Years	Marked improvement	10 mo.	5/ 1/37	450	150	0.25 Cu	0.46	31	20
				6/26/37	450	150	0.25 Cu	0.46	31	20
				2/10/38	550	200	0.5 Cu	0.95	44	20
66 48 M	1 yr.	Marked improvement	2 mo.	11/22/38	500	200	0.5 Cu	0.95	44	20
67 65 M	Years	Cure	1 yr. 6 mo.	7/27/37	500	150	0.25 Cu	0.46	31	20
68 50 F	Years	Failure	7 mo.	3/23/38	350	150	0.25 Cu	0.46	31	20
				3/30/38	250					
69 43 M	Years	Marked improvement	9 mo.	3/ 2/38	450	150	0.25 Cu	0.46	31	20
70 49 M	Years	Marked improvement	3 mo.	11/ 1/38	450	150	0.25 Cu	0.46	31	20
71 45 F	Years	Marked improvement	8 mo.	5/23/38	300	150	0.25 Cu	0.46	31	20
				6/18/38	300					
				7/29/38	300					
72 47 F	2 yr.	Marked improvement	7 mo.	3/11/38	400	150	0.25 Cu	0.46	31	20
				4/22/38	400	200	0.5 Cu	0.95	44	20
73 64 F	Years	Marked improvement	1 yr.	12/23/37	450	150	0.25 Cu	0.46	31	20
74 50 M	Years	Marked improvement	1 yr.	12/ 2/37	500	150	0.25 Cu	0.46	31	20
75 47 M	Years	Marked improvement	1 yr.	1/26/38	350	200	0.5 Cu	0.95	44	20
				2/ 1/38	400					
				4/ 4/38	400					

charge and nasal obstruction. A single small polyp was found anteriorly in the left middle meatus. When the patient was examined six weeks after her treatment no evidence of the polyp could be seen, and her symptoms had completely subsided. At the end of six months she again presented herself, with a return of her former symptoms, and on examination the nasal polyp was again found, about the same as before. The patient was retreated, and on reexamination, six weeks later, the polyp could not be found and she was again symptomless.

About the same observations were made in the case of patient 62, except that only four months elapsed between treatment and the reappearance of a solitary polyp.

I have been unable to formulate definite rules by which one might predict in advance those patients who will react favorably to roentgen therapy.

My best results were obtained in children, for whom I believe it to be the treatment of choice. (See table 2.) Marked beneficial results were obtained in mildly allergic persons, but in the severely allergic no benefits were obtained. I feel that the benefit obtained in patients with allergic rhinitis with secondary sinusitis is due to temporary inhibition of the mucous and serous glands, so that the patient is not constantly annoyed by a profuse discharge. Many of those treated remarked that they had fewer colds after the therapy.

TABLE 2.—*Analysis of the Results in Seventy-Five Patients with Chronic Sinusitis Who Were Treated with Roentgen Rays*

Age Group	Patients Treated	Patients Cured	Patients Markedly Improved	Patients Not Benefited
4 to 17 years	15	9 (60%)	3 (20%)	3 (20%)
17 to 41 years	43	5 (11%)	20 (38%)	18 (41%)
41 to 66 years	17	2 (11%)	8 (38%)	7 (41%)

Table 2 shows the age distribution of the patients in my series and the results obtained in the treatment of each group. It will be noted that the best results were obtained in the treatment of persons under 17 years of age. Of the three failures in this group one was in a severely allergic person and measles developed in 1 patient during the course of treatment, necessitating a prolonged interval between treatments. The other failure was in a 4 year old child (patient 4, table 1) who had had sinal trouble for two years. Previous to his roentgen ray therapy he had undergone tonsillectomy and adenoidectomy and a series of treatments with short wave diathermy without relief. He lived some distance away, and I was unable to examine him after his treatment. The mother reported at the end of six months that there was no relief from symptoms, but after one year she reported that during the preceding six months the boy had been in the best of health, entirely free from sinal symptoms, with no other treatment in the meantime. I classified the result of treatment in this case as a failure, although I feel that roentgen ray therapy must have been a considerable factor in the end result.

SUMMARY AND CONCLUSIONS

The results of roentgen ray treatment of 75 patients with chronic sinusitis are reported.

No definite conclusion as to the type of case in which roentgen therapy can be used with assurance of success has been reached, although I feel it to be the treatment of choice for children.

The percentage of failures in my series could have been materially reduced by omitting from the series the severely allergic patients and those with marked polypoid changes.

I have noted no harmful effects from the proper application of roentgen rays in the treatment of sinusitis. Many of the patients have been observed continuously for more than a year after treatment.

Mildly allergic persons obtained relief of nasal symptoms.

A large percentage of the patients treated reported lower susceptibility to the common cold.

There are many possibilities for this form of therapy, but much remains to be done to determine proper technic and the type of cases in which it should be used.

In 2 instances moderate-sized solitary polyps completely disappeared for a period of three to four months after treatment.

This form of treatment is not a cure-all for sinusitis but a valuable addition to the otolaryngologist's armamentarium in selected cases.

TECHNIC FOR THE REPRODUCTION OF ANATOMIC AND PATHOLOGIC MODELS IN LATEX RUBBER

AN IMPROVED METHOD

SAMUEL PELUSE, M.D.
LOS ANGELES

The technic herein described for the reproduction of anatomic and pathologic models in latex rubber includes several principles which were not included in previously described methods, and has been simplified for ready application by the postgraduate student in otorhinolaryngology.[1] The process is most applicable to those specimens which have numerous undercuts or deep crevices and cells. The model of the larynx shown in figure 1 is representative of the more complicated types and lends itself admirably to demonstration, as its reproduction embodies most of the major problems one will encounter in this technic. The reproduction of this specimen is more involved than that of simple types, but the principles applying thereto may be readily employed in the construction of practically all forms and shapes of models.

The following step by step description of the reproduction in latex rubber of the plaster of paris larynx model (fig. 1 *A*) will furnish a complete and detailed account of the process.

OUTLINE OF THE PROCESS

1. Preparing the rubber "negative" (mold).
2. Making the initial "positive" (cast) of soluble plaster.[2]
3. Constructing the plaster of paris "negative" (mold).
4. Casting the final rubber "positive."
5. Tinting and mounting the finished model.

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This method was demonstrated at the 1939 meeting of the American Academy of Otolaryngology, in a three day continuous course at the Palmer House, Chicago.

1. This article is a supplement to the article entitled "A Simplified Technic for the Construction of Life Masks of Latex Rubber," published in a previous issue of the ARCHIVES OF OTOLARYNGOLOGY (**31**:955 [June] 1940).

2. A special plaster, called Aker's new impression plaster, which contains potato starch, was used. This was furnished me by the Coe Laboratories, Chicago.

TECHNIC IN DETAIL

1. *Preparing the Rubber "Negative" (Mold).*—(*a*) Applying the Latex Rubber to the Model: Liquid latex rubber is applied with an inexpensive brush about 1 inch (2.5 cm.) wide. The bristles of the brush are first treated by dipping them into liquid soap and then gently squeezing them between the fingers to remove the excess.[3] The liquid

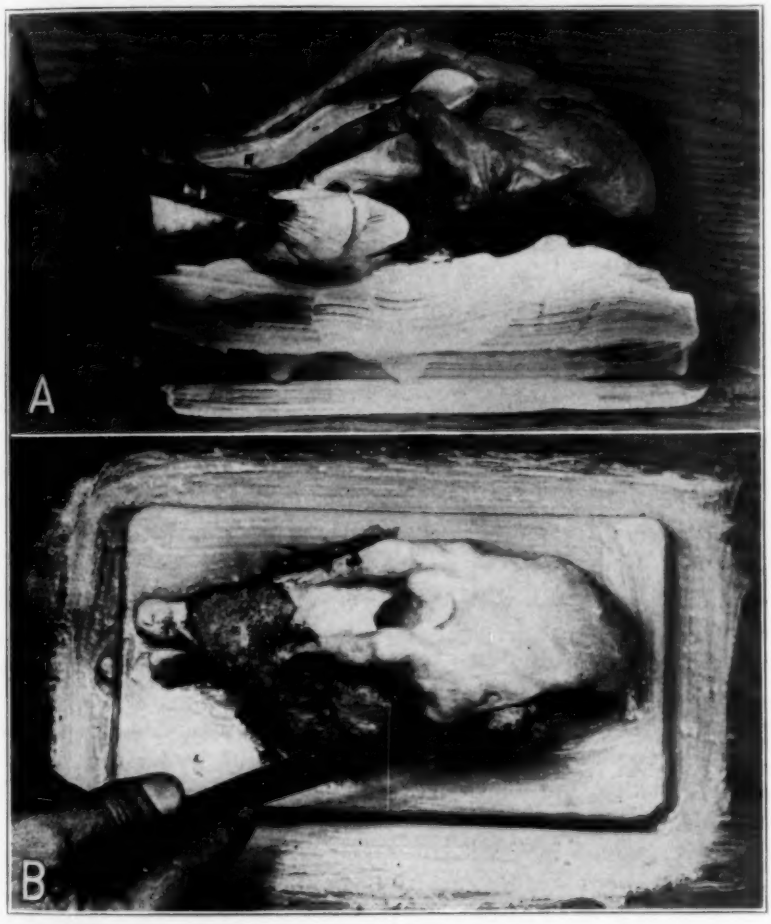

Fig. 1.—*A*, applying liquid latex rubber with a brush over a plaster of paris model of a larynx. A band about 1 inch (2.5 cm.) wide encircles the base. *B*, the larynx model covered with several layers of liquid latex rubber, which have vulcanized thoroughly. Coarse backing and filling material mixed to a paste is being applied to the undercuts and crevices with a spatula.

3. This step is essential, as it leaves a fine film of emulsion on the bristles, which prevents the rubber from adhering to the brush and thus facilitates cleansing. Brushes are cleansed in cold running water after use. If the latex has dried on the bristles, immersion in turpentine for a short while will facilitate cleansing.

latex rubber is applied by briskly brushing it over the entire surface of the model, working it into all of the crevices and undercuts until a smooth layer, devoid of air bubbles, is deposited. In addition to covering the model, a layer of liquid rubber is brushed onto the board around the base of the model, forming an encircling band approximately 1 inch (2.5 cm.) wide, as shown in figure 1 *B*. This band of rubber, when vulcanized, serves to anchor the plaster model to the board as well as to provide a support for the plaster shell, which is described later. This first layer is allowed to vulcanize (dry) by either the oven or the open air method.

If the model is constructed of a type of material that is not affected by heat, this first layer of latex rubber may be vulcanized in an oven, which is regulated to approximately 150 F. Care must be exercised at this point, as excessive heat may vaporize the water in the liquid latex, thus causing the vulcanized rubber to separate from the model.

If the model is composed of wax or of other material that will be affected by heat, then the air vulcanization method is the one of choice, and drying is readily accomplished by placing the latex-covered model in a current of air generated by an electric fan or near an open window. Vulcanization takes approximately thirty to forty minutes in the open air and less time in a regulated oven.

Successive layers of liquid latex are applied over the model and around the base (each layer being allowed to vulcanize thoroughly before the next is added) until a thickness of approximately ⅛ inch (0.32 cm.) is attained. About six or seven layers should be sufficient.

(*b*) Applying the flexible Rubber Backing and Filling Material[4]: This material is a coarse, dry, powdery substance and is mixed with liquid latex rubber until a stiff paste is formed. The mixture is then applied with a spatula (fig. 1 *B*) over the surface of the vulcanized latex rubber mold. All of the undercuts and depressions are filled in, so that the resultant surface is relatively smooth and tapers upward from the board to the top of the mold, as depicted in figure 2. The mold is then allowed to vulcanize (oven or air vulcanization) for about twelve hours, after which time it is found to be tough and resilient, with all the characteristics of a coarse rubber product.

(*c*) Building the Plaster of Paris Shell: The flexible rubber mold will not retain its shape unaided after it is separated from the larynx model. For that reason it is necessary to construct a rigid shell over the mold. The material best suited for this purpose is plaster of paris.

4. The purpose of the application of this coarse rubber material is to build up rapidly all undercuts and depressions which remain after successive coats of latex rubber have been applied, thus producing a smooth tapering surface for the subsequent plaster of paris shell. This material was supplied by the Technical Supply Company, Palo Alto, Calif.

The shell may be made in one or two pieces, depending on the irregularities of the surface to be covered. In this particular case, because of the unevenness of the surface, a two piece mold is most practical. It is constructed as follows:

Modeling clay is used to construct a wall about 1 inch (2.5 cm.) high, extending lengthwise over the top surface of the model and dividing it in half. A dam is next constructed of modeling clay, encircling one-half the base of the model at a distance of about ¾ inch (2 cm.). A thick plaster of paris [5] mixture is prepared and poured into the dam, and by rapidly shaping this preparation with the hands

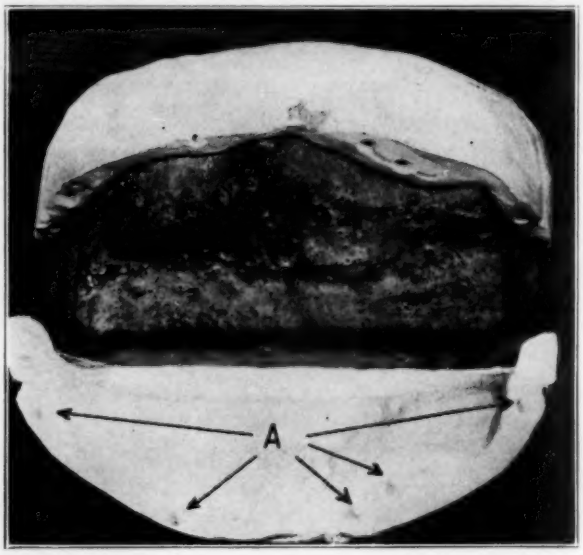

Fig. 2.—Two piece plaster of paris shell constructed over a tapering rubber mold. Note the holes in the face of the upper half of the shell and the registering "keys," *A*, in the lower half.

to a thickness of about 1 inch (2.5 cm.) over all as it is setting, half of the shell is constructed.

When this has hardened, the clay wall which runs lengthwise over the mold is removed and used to construct a dam around the base of the remaining half of the model. At this point four or five shallow holes [6] are dug out of the face of the plaster shell which has been formed by

5. A quick-setting dental plaster or any inexpensive plaster of paris will serve the purpose.

6. These holes will act as registering points for the "keys" (fig. 2 *A*), which form when the second half of the shell is cast. The number of holes is optional.

the wall of modeling clay, as shown in figure 2, in the upper half of the shell. This is accomplished by the use of an ordinary teaspoon.

A separating medium, consisting of liquid soap, is brushed thoroughly into the face of the plaster shell. Care is taken to brush the medium well into the depths of each hole.

A thick plaster of paris mixture is again prepared, poured into the dam and smoothed over all, as described previously, and the second portion of the shell is constructed. When this half is dry, the two sections are separated by inserting a screw driver in the seam and striking several light blows with a hammer.

(*d*) Separating the Rubber Mold from the Model: After the shell of plaster is removed, the rubber mold is carefully separated from the board by gently raising the edges of the encircling band. Then with careful manipulation the rubber mold is separated from the model; extra digital exertion is required when separating the mold from undercuts and deep crevices. After the mold is removed from the model, it is fitted into the plaster of paris shell, which is held together with a stout piece of cord.

2. *Making the Initial "Positive" (Cast) of Soluble Plaster.*[7]—A medium-sized rubber mixing bowl is filled about two-thirds with water, and the plaster is sprinkled rapidly over the surface until small heaps of dry plaster appear on the water. With a spatula this preparation is thoroughly mixed until a creamy consistency is secured. This mixture is poured into the mold and the undercuts and crevices are filled by brushing the material in briskly with a no. 10 camel's hair brush. When the mold is filled with this soluble plaster mixture, it is vibrated, or tapped on the sides and bottom, in order to dislodge any air which may have been trapped and to allow the plaster to flow into the smallest crevices and cracks. The surface is smoothed with a straight edge, and the cast is set aside to harden.

When the plaster has hardened sufficiently, the rubber mold is separated carefully from the resultant soluble plaster "positive" (fig. 3 *A*).[8] Any defect in this cast is repaired at once with a fresh soluble plaster mixture.

3. *Constructing the Plaster of Paris "Negative" (Mold).*—At this point a little thoughtful planning is necessary in order to construct a mold from which flawless models in rubber may be produced. A two

7. This special soluble plaster has a setting time of ten to fifteen minutes and therefore can be mixed and poured without undue haste.

8. The operator who is interested only in making plaster of paris reproductions of the original model may make any number of casts, identical to the original, as depicted in figure 3 *B*, in the mold shown in figure 3 *A*.

piece mold for this type of larynx model will cause the resultant rubber cast to show a "seam" the full length of the tongue, epiglottis and larynx, and this result is undesirable. The only apparent solution to this problem is a three piece mold, and although this type of mold

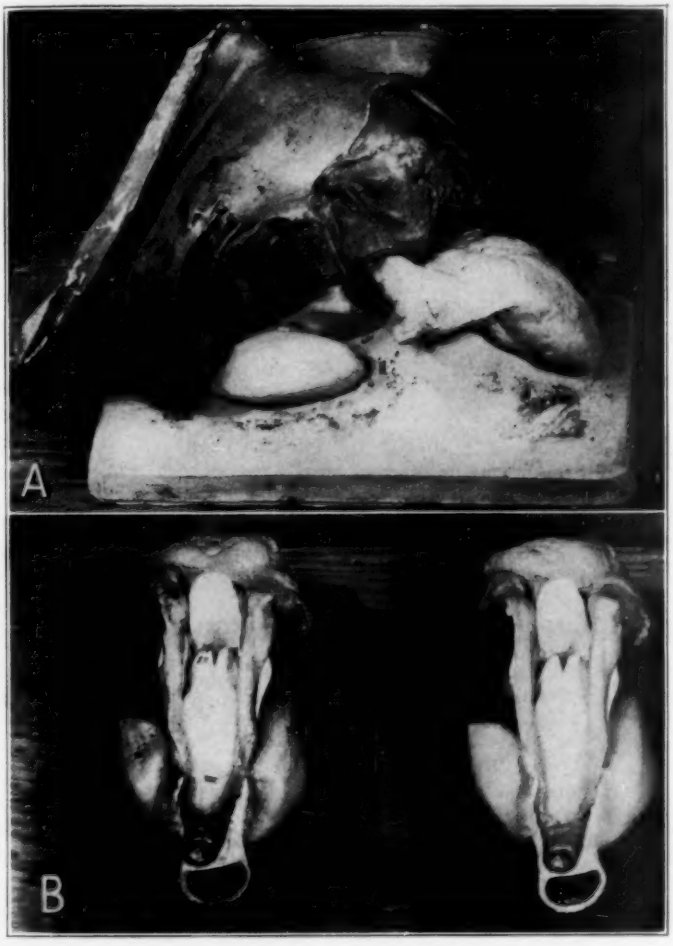

Fig. 3.—*A*, the rubber mold partially separated from the soluble plaster cast by careful manipulation. *B*, the original larynx model (left) and a tinted plaster of paris reproduction (right). The operator may produce any number of identical plaster of paris models, similar to the one shown on the right, from the model appearing in *A*.

involves an extra effort on the operator's part, the resultant "seamless" rubber cast makes the effort worth while. The following paragraphs describe one method of constructing the three piece mold.

A wall of modeling clay about 1 inch (2.5 cm.) wide is constructed along the lower edge of the tongue and along all the overhanging edges of the upper surface of the model, as depicted in figure 4. With a camel's hair brush, the whole surface of the soluble plaster "positive" which is blocked off by the clay is brushed over thoroughly with a separating medium of liquid soap.

A thick mixture of hard plaster of paris [9] is prepared in the rubber mixing bowl and poured and brushed into all of the crevices and undercuts of the upper surface of the "positive" blocked off by the clay wall. The plaster is heaped up with the hands while it is setting, until this portion of the mold has attained a thickness of approximately 1 inch (2.5 cm.). When this has dried, the clay wall is removed, and registering holes are made, as before, with a teaspoon, on each side of the faces formed by the clay.

Fig. 4.—Soluble plaster cast, shown in figure 3*A*, blocked off with modeling clay along the entire under surface of the overhanging edges, preparatory to the construction of a three piece mold.

The remaining two pieces of the mold are constructed separately. The model is divided longitudinally in half by constructing a clay wall at each end. This clay wall is anchored to the board and is then continued about the base of one half of the model, in the form of a dam. This encircling dam is constructed about ¾ inch (2 cm.) from the base of the model.

The portion of the model which is blocked off with the clay and the face of the first piece of the mold which contains the registering holes are brushed thoroughly with the separating medium of liquid soap. The board, with the model, is now propped on a support at an angle of approximately 30 degrees, in order to facilitate the flow of the plaster of paris into the undercuts and crevices. A thick plaster of paris mix-

9. A hard plaster of paris called Coecal (setting time ten minutes), furnished by the Coe Laboratories, was used.

ture is again prepared and is brushed and poured into the farthest recesses of the blocked-off area. While this is setting, it is molded to a thickness of approximately 1 inch (2.5 cm.) over all with the hands and is smoothed down until the top surface is on the same plane as the edge of the first piece cast. When this has set, the clay dam is removed, and a similar one is constructed around the base of the remaining half of the soluble plaster cast.

The third piece of the mold is prepared and cast in a corresponding manner and is then set aside to harden.

When the plaster of paris mold is set, the combined structure, consisting of the soluble plaster "positive" and the plaster of paris "negative," is placed in a deep basin of cold water, so that the surface of the water is about 1 inch (2.5 cm.) above the top of the plaster of paris. The water is slowly brought to a boil and allowed to boil actively until all of the soluble plaster has disintegrated, leaving the three pieces of the plaster of paris mold separated and devoid of debris. However, if any soluble plaster remains adherent to the plaster of paris mold, it is easily removed by allowing a stream of hot water from the tap to play over it, and the attached particles are brushed away with a camel's hair brush. A sharp instrument is not to be used, as there is danger of scratching the surface of the mold.

The three pieces of the mold are reviewed at once, and any damaged areas or air pockets are corrected with a fresh plaster of paris mixture. The three pieces of the mold are then set aside to dry thoroughly. An oven regulated to 150 F. may be used.

4. *Casting the Final Rubber "Positive."*—When the three sections of the mold have dried thoroughly, they are assembled and held together with a stout cord. A small amount of liquid latex rubber is poured into the depths of the mold and brushed thoroughly into the crevices with a brush whose bristles have been treated with liquid soap as previously described. This first layer is brushed briskly over the face of the entire mold, as well as the encircling shelf of plaster which was formed by the clay dam during the casting process. This layer is allowed to vulcanize thoroughly, and then successive layers are applied, with the precaution that each layer is allowed to vulcanize completely before the next layer is applied. After the sixth or seventh layer has been applied, a full thickness of gauze (8 ply) is laid over the entire surface of the mold, and liquid latex rubber is brushed thoroughly into the meshes. When vulcanized, this rubber impregnated gauze acts as a rigid reenforcement to the resultant model. The rubber cast has attained a thickness of approximately ⅛ inch (0.30 cm.) at this time and is ready to be separated from the mold.

The stout cord used to assemble the three piece mold is removed first. Then the rubber is peeled away from the encircling shelf of

plaster and from the surface of the mold. One of the lower sections of the three piece mold is next gently manipulated and maneuvered until it is separated and free of the rubber cast. The top piece of the mold is then loosened and gently separated from the rubber cast. This maneuver

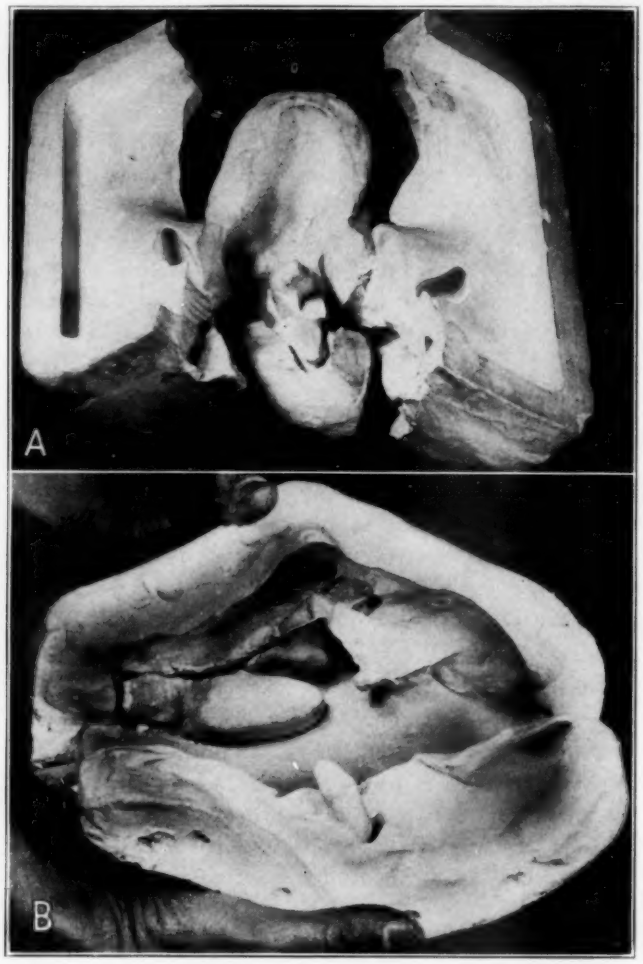

Fig. 5.—*A*, three piece mold. The three sections are shown as they appear after the soluble plaster has been boiled and brushed away. *B*, removing the rubber cast from the mold. One section of the mold has already been removed and does not appear here. The top section is in the operator's right hand and the third section in the left hand. A part of the rubber cast is visible.

must be carefully executed in order to avoid breaking off any projecting parts of the mold (fig. 5 *B*). The third section of the mold is separated from the rubber cast, and the "seams" are trimmed away with scissors.

The encircling band of rubber around the base is trimmed away, and the model is now ready for tinting and mounting.

5. *Tinting and Mounting the Finished Model.*—I find it expedient to tint these rubber models with an air brush and special rubber lacquers. However, excellent tinting has been produced on these models by the postgraduate students, with a fine camel's hair brush and india inks (Higgins) of various colors, namely, blue, red, white and black. Mounting of this model after it is tinted is optional. Figure 6 shows the larynx model, with its base trimmed away, mounted on a board with three other rubber models. This board, with the four models, was exhibited at the meeting of the American Academy of Otolaryngology in Chicago, in 1939..

Fig. 6.—Rubber larynx model mounted on an exhibition board with other rubber reproductions (hematoma of the left ear, hump nose and prosthesis for an exenterated left orbit).

Figure 7 shows the practical application of this type of model for instructing postgraduate students and interns in the technic of mirror

laryngoscopy. This larynx model has been ingeniously mounted on a home-made bell-ringing device by one of the postgraduate students in the department of otology, rhinology and laryngology of the University of Illinois College of Medicine. It consists of an ordinary cigar box, into which a bell and a 2 volt battery have been placed. These, in turn, are wired to a contact switch on the cover of the cigar box. The points of the contact switch are wired to the various parts of the larynx and are labeled accordingly as follows: right arytenoid, right vocal cord, right piriform sinus and left piriform sinus, left vocal cord and left arytenoid, respectively, reading from the operator's left to his right.

Fig. 7.—The rubber larynx model mounted on a home-made bell-ringing device, as it is used for instructing postgraduate students and interns in the technic of mirror laryngoscopy. The lamp is the author's patented portable light, for use with a head mirror.

The circuit is closed with the laryngeal applicator; this causes the bell to ring.

The lamp is the author's patented portable light and is employed in the department for use with a head mirror.

SUMMARY AND COMMENT

A practical technic for the reproduction of anatomic and pathologic models in latex rubber is described.

These rubber models are ideal for instructional purposes. They are relatively inexpensive to construct and are unbreakable.

The unlimited number of models which can be reproduced in each mold assures each student an individual specimen for purposes of study or for building up a permanent collection.

This type of work is an important part of postgraduate study in otorhinolaryngology in many universities, and the described practical technic will facilitate and encourage the reproduction of models on a larger scale.

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RHYTHMIC STIMULATION OF THE LABYRINTH

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Among the various methods of stimulating the labyrinth, electric stimulation is rather neglected, apparently because it acts not only on the receptor organ but also on the afferent neuron (ganglion vestibulare, Dohlman [1]). The possibilities of electric stimulation, however, appear not to have been completely exhausted, since generally only stimulation with a constant (direct) current is used, stimulation with faradic current yielding no definite reactions (Klestadt [2]). The ineffectiveness of the faradic current, however, does not exclude the possibility that the labyrinth might react on rhythmic stimulation with frequencies lower than those produced by the usual faradic apparatus. It seemed, therefore, worth while ascertaining how the labyrinth [3] might react to rhythmic electric stimulation with various frequencies.

METHOD

The following arrangement (fig. 1) was used in experiments performed on cats: Circuit 1 consisted of a dry cell or storage battery, the coil of an electromagnet, a vibrating spring of variable length with a platinum wire on the end dipping into mercury (the vibrating interrupter of the Harvard Instrument Company). The magnet of a sensitive relay was connected in parallel with this circuit, so that the relay was actuated in accordance with the vibrations of the spring.

There was also shunted across the contact points a condenser, which reduced contact sparking and insured continuous operation of the interrupter. The stimulating current was interrupted by the relay (R), which formed with the stimulating electrodes circuit 2. The source of the stimulating current was a direct current of variable voltage, so that currents of from 1 to 10 milliamperes could flow through the labyrinth. For comparison faradic current (Harvard inductorium) and sinusoidal current (60 cycles, 2 to 15 volts) were also employed.

Read before the Philadelphia Neurological Society, May 24, 1940.

From the Department of Experimental Neurology (D. J. McCarthy Foundation) of the Temple University School of Medicine.

1. Dohlman, G.: Experimentelle Untersuchungen über die galvanische Vestibularisreaktion, Acta oto-laryng., 1929, supp. 8, p. 1.

2. Klestadt, W., in Bumke, O., and Foerster, O.: Handbuch der Neurologie, Berlin, Julius Springer, 1936, vol. 4, p. 359.

3. The term "labyrinth" as used here covers not only the receptor cells of the peripheral sense organ but also the afferent neuron. The exact site of action of the rhythmic stimulation will be studied in further experiments.

The stimulating electrodes consisted at first of two metal tubes placed in the external auditory canals, so that the current passed transversely through the skull. For monaural stimulation one electrode was introduced into the external canal, and a plate electrode was fastened on the abdomen. With these types of stimulation, a spread of the current to neighboring muscles and nerves could hardly be avoided. To procure as nearly as possible isolated stimulation of the labyrinth, an electrode of the concentric needle type was constructed. It was made up of a thin metal tube, insulated on its external surface and containing a wire that was insulated except at its protruding tip. After the bulla ossea had been opened, the tip of the electrode was either introduced into the round window or placed in a shallow groove that was chiseled into the promontory. The electrode was rigidly fixed on the head holder so that its tip remained in the inner ear after the head was turned back to its normal position. This method of stimulation had the advantage that a spread of the current, e. g., to the facial nerve, was avoided and 1 to 3 milliamperes was sufficient to produce reflex reactions of the eyeballs, whereas if the electrodes were introduced into the external meatus currents up to 9 milliamperes were necessary. By exercising care in placing the tip of the electrode in the round window or in

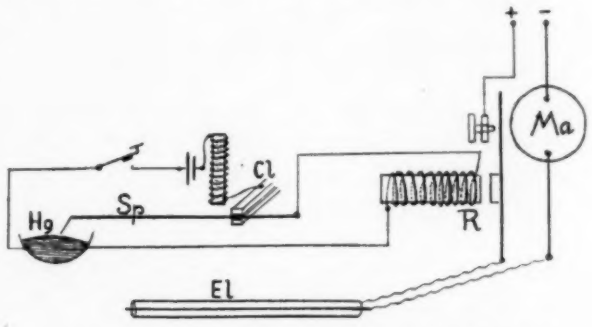

Fig. 1.—Apparatus for rhythmic stimulation of the labyrinth. *Cl* indicates a clamp; *El*, the concentric needle electrode; *Hg*, mercury; *R*, relay; *Ma*, the milliampere meter, and *Sp* a spring.

the shallow groove on the promontory, the appearance of spontaneous nystagmus could be avoided. If injury to the inner ear produced spontaneous nystagmus, electric stimulation was often effective in suppressing it. If spontaneous movements of the eyeballs persisted, they could be distinguished without difficulty from the reactions to the electric stimulation, as later explanation will show.

The rate of stimulation was recorded by connecting a string galvanometer parallel to the stimulation circuit; the resistance in the circuit of the string galvanometer was kept so high that stimulation of the ear was practically unaffected, as was shown by control experiments, in which the string galvanometer was disconnected. In order to record the movements of the eyeballs the method described by Majewski [4] was used in a simplified modification. A small rubber cap,[5] 3 mm. in diameter, was pressed on the anesthetized cornea (usually of the right eye).

4. Majewski, K.: Eine neue Methode der klinische Nystagmographie, Arch. f. Ophth. **96**:140, 1918.

5. A so-called rubber policeman (Eimer & Amend), used by chemists for scraping precipitates from the side of beakers.

This cap was compressed by a forceps before application, and the pressure was released after the cap had been placed on the cornea, so that it was held in place by the suction of the vacuum in its interior. The shadow of a thin needle sticking in the cap was projected on the recording paper by the lamp of the string galvanometer. In order to prevent interference from movements of the lids, these were kept apart by an eye speculum. The reactions of the eyes were also directly observed without recording.

RESULTS AND COMMENT

With low frequencies of stimulation the reactions still show characteristics such as those induced by galvanic stimulation. One or more eye jerks appear while the current is on and may be followed by a reaction with each break of the current. The ocular movements are of a mixed type, horizontal and rotary. They also often have a vertical component. During the flow of the current the fast component of the eye jerks is directed toward the cathode, while during the periods when the current is broken the direction of the ocular reactions is less constant (in some cases toward the cathode, in others toward the anode). Thus on stimulation with low frequencies (1 to 5 per second, sometimes even with a somewhat higher rate) not only a nystagmus which follows the rate of stimulation may appear, but also a nystagmus with a multiple frequency (fig. 2 *A*). If the frequency of stimulation is further increased, this difference between the rhythm of stimulation and that of the nystagmus is no longer found, each stimulus eliciting one jerk only. A fast and a slow component of the nystagmic movements of the eyeballs could be distinguished only at frequencies below 10 per second (fig. 2 *B*). At higher frequencies the reaction assumed the characteristics of a tremor (fig. 2 *C*), the amplitude of which in general decreased with increasing frequency (fig. 2 *C* and *D*). One could sometimes observe, however, spontaneous, more or less regular fluctuations in the amplitude of the reaction (fig. 2 *G*); these fluctuations probably are related to fluctuations in the excitability of the vestibulo-ocular reflex arc. It may be recalled in this connection that the spontaneous nystagmus after unilateral labyrinthectomy and also Bechterew's compensatory nystagmus show a periodic increase and decrease of the ocular reactions (Spiegel and Yaskin [6]). A tendency toward periodicity of the reactions could also be found on continuous caloric or galvanic stimulation of the labyrinth (Spiegel and Aronson [7]). In some graphs the eye jerks produced by the rhythmic electric stimulation were superimposed on spontaneous volitional movements or on a spontaneous nystagmus of lower

6. Spiegel, E. A., and Yaskin, J.: Experimental-Studien am Nervensystem, Ztschr. f. d. ges. exper. Med. **63**:515, 1928.

7. Spiegel, E. A., and Aronson, L.: Continuous Stimulation of Labyrinth with Sustained Nystagmus, Arch. Otolaryng. **17**:311 (March) 1933.

frequency (fig. 2 *E* and *F*). A spontaneous nystagmus produced by opening of the inner ear could, however, be suppressed or masked by the reaction to rhythmic electric stimulation.

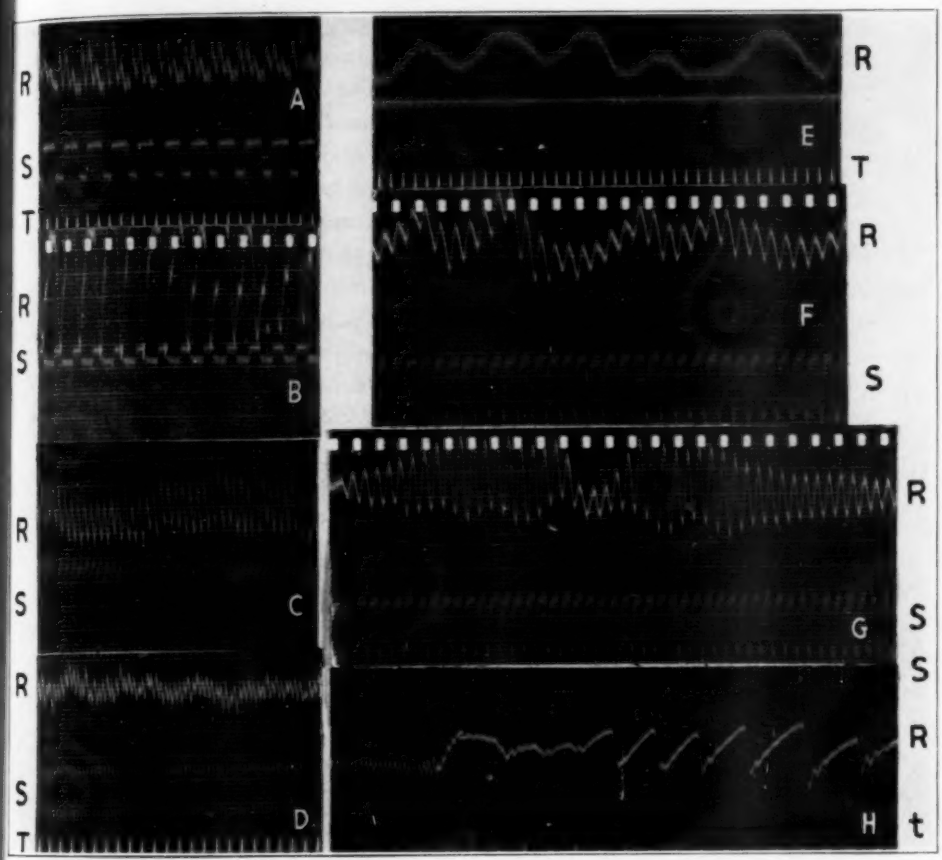

Fig. 2.—Ocular reactions to rhythmic stimulation of the labyrinth. *R* is an optical record of the movements of the right eye (details described in the text); *S* indicates the stimulation of the left labyrinth (fluctuations of the stimulating current are recorded by a string galvanometer); *T*, intervals of one-tenth second; *t*, intervals of one-fifth second. (The recording paper was moved at the same speed in all experiments.) *A*, stimulation at the rate of 4 per second is answered by 12 jerks per second. *B*, stimulation at the rate of 6 per second evokes nystagmus with slow and fast components at the same rate. *C*, stimulation at the rate of 17 per second. *D*, stimulation at the rate of 27 per second. The ocular reactions in *C* and *D* are tremors in the frequencies of the stimulation. *E*, spontaneous nystagmus (84 per minute) after opening of the left labyrinth. *F*, ocular jerks in the rhythm of stimulation (9 per second) superimposed on the spontaneous nystagmus. *G*, spontaneous fluctuations in the amplitude of response (rate of stimulation 11 per second). *H*, after-discharge following stimulations at the rate of 25 per second.

In attempting to interpret these experiments one may raise the question whether the reactions to rhythmic stimulation which are recorded here are due to excitation of the vestibulo-ocular reflex arc or only to direct stimulation of the ocular muscles or their motor neurons. A reflex effect is suggested by the fact that synergic reactions of both eyes can be elicited by stimulation of the labyrinth with a concentric needle electrode, avoiding a spread of the current to neighboring nerves, the facial, for example. Furthermore, an excitation of the reflex arc is indicated by the appearance of an after-discharge when the rhythmic stimulation ceases. Figure *2 H* shows a graph resulting from such an experiment. About one second after the stimulation, at a frequency of 25 per second, is discontinued, nystagmus of lower frequency (3 per second) and larger amplitude appears. This seems to indicate that an excitatory state is built up in the reflex center.

Finally, the connections between one labyrinth and the nuclei of the ocular muscles were severed by an incision on the inner side of the restiform body (fig. 3), and the concentric needle electrode was inserted

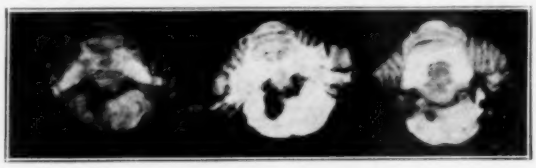

Fig. 3.—Incision on the inner side of the restiform body.

into the homolateral labyrinth. Rhythmic electric stimulation of this labyrinth failed to produce ocular reactions even if the current was so increased that twitches of the facial muscles, due to a spread to both facial nerves, appeared.

The highest frequency of stimulation which yielded *distinct* eye jerks in the rate of stimulation was 27 per second. At higher frequencies the ocular reactions observed were not definite enough to be considered positive, or a fine tremor appeared, which often had a lower frequency than the stimulation. In one cat, for instance, stimulation at a rate of 65 per second induced a tremor of 17 to 23 per second. The fact that distinct reactions to rhythmic stimulation of the labyrinth can be produced only at relatively low frequencies probably is related to the relatively high chronaxia (Bourguignon,[8] Vogel,[9] Altenburger,[10] Dohlman [11]) of the vestibular nerve.

8. Bourguignon, G., and Déjean, R.: Chronaxie normale du nerf vestibulaire de l'homme, Rev. neurol. **1**:1017, 1927; Les syndromes chronaxiques et le rôle de la chronaxie dans le diagnostic des maladies nerveuses centrales et périphériques, Paris méd. **2**:279, 1931.

Rates of stimulation which were apparently inadequate to produce definite ocular reactions by acting on the vestibulo-ocular reflex arc did, however, sometimes influence nystagmus caused by injury to the inner ear. The application of such stimuli in some instances increased the frequency and amplitude of a weak "spontaneous" nystagmus; in others a latent nystagmus became manifest. A similar effect was obtained by stimulation with a rather intense faradic current (e. g., distance of the coils, 8 cm.) or with a sinusoidal current of 60 cycles. Thus if the left labyrinth was somewhat injured by the introduction of the concentric needle electrode, the rhythmic stimulation at relatively high frequency was followed by the appearance or an increase of spontaneous horizontal and rotary nystagmus to the right. This was sometimes preceded by coarse nystagmus to the stimulated labyrinth (in this case to the left, with a component upward). In analyzing the effects of the experiments described here one must bear in mind that rather strong currents had to be applied in order to produce the reactions recorded. Control experiments showed that strong stimulation of sensory nerves, such as the sciatic, can also increase the amplitude and frequency of an existing weak spontaneous nystagmus. Irradiation of impulses from other sensory nerves and pathways to the vestibulo-ocular reflex arc seems therefore to play an important part in the genesis of the effects described here on spontaneous nystagmus.

SUMMARY

While it is generally assumed that electric stimulation of the labyrinth yields reactions only if direct (constant) current is employed, it was shown by experiments on cats that the labyrinth responds to a rhythmically interrupted current. Binaural stimulation was used (one electrode in the external meatus of each ear) as well as monaural stimulation by a concentric needle electrode that was introduced into the inner ear through the bulla ossea. The latter method required smaller currents (1 to 3 milliamperes) than the former (up to 9 milliamperes) to

9. Vogel, P.: Beiträge zur Physiologie des vestibulären Systems beim Menschen, Arch. f. d. ges. Physiol. **230**:16, 1932.

10. Altenburger, E.: Beiträge zur Vestibularis-Chronaxie, Ztschr. f. d. ges. Neurol. u. Psychiat. **138**:332, 1932.

11. Dohlman, G.: Mechanism of Transformation into Nystagmus on Stimulation of the Semicircular Canals, Acta oto-laryng. **26**:425, 1938. Dohlman claimed that he obtained slight deviations of the eyes synchronously with stimulation at frequencies over 150 per second. It seems, however, doubtful whether the exceedingly small movements which he recorded were true vestibulo-ocular reflexes. He himself emphasized the fact that they occurred at a much lower intensity than that necessary for evoking nystagmus and even at a lower level than that required to produce deviation of the eyes.

elicit nystagmic responses of the eyeballs. With a low frequency of stimulation (1 to 5 per second) the nystagmus may show a frequency higher than that of the stimulation, reactions appearing while the current is on as well as with each break of the current. With a higher rate (6 stimuli or more per second) the rhythm of the nystagmus corresponds to that of the stimulation. A slow and a fast component may be distinguished at rates below 10 per second, while at higher rates the reaction assumes the characteristics of a fine tremor, which may be followed by a coarse nystagmus after the stimulation ceases. Definite ocular reactions in the rhythm of the stimulation were observed at frequencies up to 27 per second.

MECHANISM AND TREATMENT OF NEUROTIC SYMPTOMS ESPECIALLY OF THE EAR, NOSE AND THROAT

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Generally speaking, one may say that psychoneurotic symptoms result from emotional disturbances. These emotional disturbances may be relatively acute in an otherwise stable person, or, as is so often the case, they stem from a chronic, lifelong instability of personality. They are translated into symptoms by means of: (1) tension primarily in the autonomic nervous system and (2) symbolism. When a person is afraid, or hates or is irritable, his heart rate tends to increase, his blood pressure to rise, his intestines to contract. These manifestations result from disturbed activity of the autonomic nervous system (usually both the sympathetic and the parasympathetic systems are involved), and since every portion of the organism is under the influence of these symptoms, the unstable and emotional person tends to have symptoms in most of the body. Many neurotic patients will come to the physician with one presenting complaint, yet on more thorough investigation will be found to have many other symptoms. These symptoms are symptoms of tension. On the other hand, ideational disturbances tend to take the form of disturbed symbolic representations and utilize such psychologic mechanisms as identification, rationalization, dissociation and projection. All psychoneurotic symptoms may be spoken of in terms of either tension (in the central and autonomic nervous systems) or symbolism. In order to understand local neurotic symptoms of any portion of the body it is necessary to understand the general nature of the neurotic symptoms.

SYMPTOMS OF TENSION

Symptoms of General Tension.—The symptoms presented by the patients suffering from psychoneuroses may be those of a state of general "psychologic" tension (stretched, overly active) or of focal "physiologic" tension of the autonomic nervous and other related systems. The tense attitudes show themselves in irritability, states of anxiety, "worry," overconcern about one's self, egocentricity and even inability to con-

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centrate on anything unrelated to one's problem. These symptoms have their basis in emotional factors, and they tend to be present, in varying degrees of intensity, in all the forms of neurosis. Such general signs and symptoms of tension are present, however, not only in patients who are neurotic but in normal persons when they are laboring under strong emotional stress. It is far more informative and illuminating to describe specific symptoms as to mechanism and locality than it is to attempt to label and to categorize the patient.

Symptoms of Focal Tension.—In addition to the general state of tension which manifests itself in the emotional attitude of the person, there are focal expressions of tension. Tension disturbances of the central nervous system may show themselves in tension of striated muscles, and overactivity of endocrine glands may be manifest through increased basal metabolism, hyperfunction of the adrenals and other conditions. Generally, however, neurotic symptoms are expressions of overactivity of the autonomic nervous system. Speaking more succinctly, one may say: Though all integrative systems are to some degree involved in tension, most focal disturbances result directly or indirectly from activity of the autonomic nervous system.

As a consequence of such focal tensions, almost every portion of the body may be involved. There may be disturbances in cardiac rate and rhythm, showing clinically as tachycardia, extrasystoles, cardiac pain resulting from spasm of the coronary vessels; there may be respiratory disturbances, expressed by deep sighs, a sense of oppression across the chest, pains in the chest, glottic spasms; there may be gastrointestinal disturbances, such as hyperchlorhydria, spasms of the cardiac or pyloric sphincters of the stomach, vomiting, spasms of the intestine and/or of the ducts of the gallbladder, mucous colitis, constipation, diarrhea; there may be disturbances of the bladder, such as frequency of urination, urgency, retention; there may be sexual difficulties: impotence, priapism, dysmenorrhea, delayed or too frequent menstrual flow; there may be abnormal perspiration of the palms and the soles or coldness of the hands and feet.

Likewise, the eyes, ears, nose and throat may be involved in tension, giving rise to such symptoms as amblyopia, tubular vision, eyestrain; tinnitus, roaring sounds, an audible pulse, exaggerated auditory acuity with startling at the faintest noise; rhinorrhea, pain, twitchings, sensation of choking or a "lump" in the throat, impeded swallowing, stammering and stuttering. All these and many more symptoms for which no definite organic pathologic basis has been discovered may result directly from overactivity of the autonomic nervous system, or indirectly, via the influence of this system on the adrenal, pituitary and other endocrine glands. The fact that the autonomic nervous system

is involved in all physiologic activity accounts for the widespread character of the symptoms in the neuroses.

These general and focal symptoms of tension occur in most psychoneurotic patients: preponderantly in those suffering from neurasthenia and states of anxiety, relatively less in those whose condition is categorized under the labels of hysteria and obsessive-compulsive neurosis, in which states symbolic symptoms are more common.

SYMBOLIC SYMPTOMS

Many of the symptoms (reaction patterns) of neurotic patients are symbolic; that is, the symptoms represent, in condensed and often obscure form, underlying emotional difficulties and conflicts. The symbol as an idea does not necessarily involve tension, though usually symptoms of tension coexist. A symbol is anything which specifically represents an idea or a group of ideas. A flag, for example, is the symbol of a country's ideas and ideals; yet many persons may come to venerate the symbol itself, even when it is used in such fashion as to violate the very ideals which it was intended to represent. In the same way, a patient may have a hysterical aphonia as a symbol of an underlying emotional conflict, and yet so concentrated is all his attention on his symptom (symbol) that he completely disregards its underlying meaning. The hysterical aphonia is a symbol, a symbol which represents unconsciously to the patient underlying disturbing emotions. Moreover, the symptom enables the patient to focus his general unhappiness and distress on a concrete and tangible illness. This finding of an outlet for the pent-up internal emotions serves a function of release, expression and the "taking one's mind off" the other and causative conditions.

REPORT OF A CASE

Mr. J. K., aged 26, was referred to the psychiatric dispensary because of hysterical aphonia. He spoke in a whispered voice and complained bitterly of his lot. The patient lived with his father behind a small store where his father sold groceries. The father was a penurious person and complained constantly about the cruel way in which life had treated him. His wife had died many years before, and he had two children, the older of whom, a daughter, had married. This daughter had seemingly forgotten completely about her father, and he constantly bemoaned the fact that his children did not care for him. He was resolved that his son should aid him and imposed the most menial tasks without compensation. The son was forced to work early and late, and after he had completed the second year of high school was kept home for this purpose. He had always had ambitions of becoming an educated and professional man, and not only were his opportunities thwarted, but he was stunted in every other way by his constant association with his nagging, complaining, irritating father. Several times he had run away, but lack of funds and failure to procure a position had forced him to return, and after each return his life was made more unbearable by his father.

One day, after a great deal of abuse, the patient called his father obscene names and fled the home, only to return late at night. He was received in cold silence, and life continued as it had till late the following night, when the father had an apoplectic stroke, followed by left-sided hemiparesis. The father had been ill for some time and had visited clinics for treatment for his "high blood pressure," but he accused his son of producing "the stroke," and the son believed that he was guilty. He could find nothing to say in reply to his father's accusations and the next day found that he could not say anything to any one except in a hoarse whisper.

The inability to speak was a symbolic manner of expressing the patient's emotional shock and feeling of guilt. The patient felt a deep resentment over the way he had been treated by his father; yet so guilty did he feel over the "crime" of having produced his father's paralysis that "he could not tell" of the relatively inconsequential wrongs his father had done to him. This "inability to tell" was symbolically expressed as the symptom aphonia. However, associated with this symbolic complaint were many other symptoms of general and focal tension. Both the symbolic symptoms and the symptoms of tension resulted from a disturbance of personality due to environmental pressure.

Many other symptoms are of symbolic nature: hysterical deafness; tubular vision; hysterical paralysis of the arms and legs; astasia-abasia, or inability to walk because of weakness in the legs (although there is excellent muscle power while the patient lies in bed); obsessive ideas which cannot be eliminated from one's thought, such as fears and peculiar ideas; compulsive actions, such as continuous hand washing or the compulsion to touch some object; hallucinations and delusions. These are but a few of the symbolic representations of underlying difficulties. Each one of the symptoms is meaningless in itself, but it has a great deal of meaning if understood as being symbolic. Such an understanding is essential for a dynamic comprehension of human activity.

It is difficult to differentiate in a clearcut fashion between symptoms of tension and symbolic symptoms, for in any given patient both types of symptoms are so intermingled that the task becomes one of separating the yolk from the white of a scrambled egg. Moreover, any given symptom may be at one and the same time an expression of tension and overactivity of the autonomic nervous system and also a symbol of some underlying difficulty. Thus one young woman complained bitterly of blushing spells, which came on so frequently that she had become desperate. Since blushing is produced by vasodilatation in the face and neck, it must be assumed to be a symptom of tension, a symptom of disturbed regulation of the autonomic nervous system. However, a further analysis of her history revealed that this young woman, married four years, had discovered her husband's infidelity, the woman in the case being her own sister. "It made her blush to think of it" besides making her "furiously angry." The blushing was thus also

a symbolic representation of her emotional conflict over the entire sexual episode. In the accompanying table, therefore, each symptom must be regarded not as belonging exclusively to one group but as being primarily of that group.

Such a classification of symptoms obviates the necessity of using the traditional, but often confusing, categories of (1) hysteria, (2) anxiety neurosis, (3) neurasthenia and (4) obsessive-compulsive neurosis. Moreover, by the use of such terms as "tension" and "symbolism" the physician is able to impress the patient with the significance of his illness and, by the very terms of the description, to suggest the method of cure.

A Few Representative Types of Neurotic Symptoms *

Symptoms Usually Symbolic but not Exclusively So	Symptoms Expressive of General Tension	Symptoms Usually Expressive of Focal Tension, i. e., of a Disturbance of the Autonomic Nervous System
Muscle paralysis	Irritability	Eyestrain
Muscle tics	Anxiety	"Ringing in the ears" (tinnitus)
Anesthesias	Egocentricity	Stammering and stuttering
Aphonia	Difficulty in concentration	"Lump" in the throat
Deafness	Worry	Difficulty in swallowing
Tubular vision	Overconcern about the self	Pain in the throat
Phobias and fears	Anorexia	Rhinorrhea
Obsessions, compulsions	Insomnia	Headache
Abnormal sex interests		Abnormal perspiration of palms
Impotence		Coldness of the hands and feet
Hallucinations		Weakness, "run down" condition
Delusions		Easy fatigability
		Pains and aches in the limbs
		Cardiac symptoms
		Gastric difficulties
		Intestinal spasms
		Irritability of the bladder
		Uterine dysfunction †

* Presuming that there is no organic disease.

† It must again be emphasized that many of the symptoms in this table are an inseparable admixture of tension and symbolism.

NEUROTIC SYMPTOMS OF THE EAR, NOSE AND THROAT

There may be neurotic symptoms related to the ear, such as ringing and buzzing, pain in the external meatus or "inside" or over the mastoid, partial or complete deafness and such involvement of the vestibular apparatus as dizziness and giddiness. Since all these symptoms are usually manifestations of definite pathologic conditions, and since it is so difficult actually to see the pathologic process, it becomes obvious that the diagnosis of neurosis must be approached with care. Yet when all symptoms point to such a diagnosis, it is an error, which may have unfortunate consequences, to insist that the ailment is of organic nature. These symptoms are generally in the nature of symptoms of tension; the pounding in the ear of which so many patients complain may result from the acute awareness by the patient of the arterial pulse in the vessels associated with the ear. The small tympanic bones are probably so tensed that sounds are more easily and quickly perceived

than by normal persons, and consequently these patients tend to startle easily. The ringing and buzzing in the ear may be partially explained on this basis, plus that of changes in the middle ear, resulting from blocking of the eustachian tube by spastic muscular contraction and an angioneurotic-like swelling of the lining cells. Pain may be more definitely associated with some minor, but actual, organic disease, which becomes intensified by the emotional state of the patient but, on the other hand, such a phenomenon as deafness may be a symbolic manifestation of underlying conflict. One patient, for example, was so upset by the continuous, shrill nagging of his wife that he suddenly discovered he could no longer hear. This inability to hear served both symbolically to express his resentment and to provide an excellent method of escape from the situation. Usually such deafness is partial and may be a factor also in the common "wandering of attention."

The nose is often the site of such neurotic symptoms as pain, peculiar paresthesias, rhinorrhea and twitching. These, too, have the same etiologic significance, namely, that of a basic emotional disturbance. The pain referred to previously often has its basis in some actual, though minor, pathologic process which has become exaggerated by the high state of tension of the patient. Emotion can bring about such changes in the physiology of the vasomotor system of the skin and membranes as to result in paresthesia. Rhinorrhea, while it may be caused by an allergic condition, can result from tension, and the causation of the secretion of the nasal mucous membranes does not differ essentially from that of the excessive secretion of the sweat glands in the palms or the hypersecretion of mucus in mucous colitis. In many instances the physician finds some minor organic cause of the rhinorrhea, but in themselves such causes are usually inadequate to explain the marked discharge. The explanation lies in the well known susceptibility of the membranes to emotional tension, so that minor alterations are sufficient to start the flow of watery discharge. Twitching of the nose generally results from subjective experiencing of paresthesias or pruritis and an attempt to assuage this feeling by puckering of the nostrils. Such twitching can also be on a symbolic basis: In the schizophrenic patient, for example, the peculiar *Schnautzkrampf* is symbolic of a primitive type of response.

The nose and throat are more often affected by neurosis than is the ear. Most common are the complaints that there is nasal blockage or a "lump in the throat" (esophageal spasm). This "lump" occasionally interferes with swallowing or with breathing, so as to cause a choking sensation, but usually it is in the nature of a feeling of constriction or of the presence of a foreign body. Examinations generally give negative results, and usually the symptom is associated with other general neurotic symptoms. The "lump" is a symptom of tension, probably result-

ing from spasm of the smooth and striated musculature in the throat, pharynx and larynx. The difficulty in swallowing is undoubtedly the result of muscular spasms. Pain in the throat occurring either after some surgical procedure or after swallowing a sharp object, such as the familiar fishbone, is often the result of a neurotic condition or state of tension. In these instances, again, there is usually some minor cause for the pain, and, in addition, an overwhelmingly preponderant state of tension, which causes the organism to exaggerate and perpetuate the original symptoms out of all proportion to the cause as reported by the patient.

Speech is commonly involved. There may be aphonia, whispered voice, hoarseness or stammering and stuttering. In whispered voice there is a greater or lesser degree of failure in the approximation of the vocal cords in the attempt at phonation. Aphonia of the whispered voice type tends to be a symptom primarily of tension and is usually associated with other physical and psychologic symptoms of tension. On the other hand, complete aphonia tends to be more symbolic, a "wish not to speak" or a "fear of speaking."

STAMMERING AND STUTTERING

Stammering and stuttering are extremely common expressions of tension. Speech is so commonly involved because tension is such a common characteristic of present day society and because speech, being one of the latest phylogenetically acquired characteristics, is therefore one of the first functions of the organisms to be disturbed under conditions of stress. There are four general types of stuttering as related to states of tension (there is only an artificial difference between stammering and stuttering), but they are not usually found in pure form. The average patient utilizes several forms, forms which differ not in causation or in mechanism but simply in the particular muscles involved. Illustrative of the first form is the difficulty of those patients who start to speak and then hold their breath before emitting a sound, which when uttered usually comes forth in perfectly formed words. In these patients the tension of the respiratory muscles prevents the relaxation and the release of air normally used in speaking. The stutterers whose speech difficulty is of the second type make many facial grimaces before vocalizing, and they may contract the muscles of the mouth, project and withdraw the tongue, wrinkle the face and forehead, before uttering a word, which when uttered may be perfectly pronounced or may be hesitant. The tension of the muscles involved in this grimacing is evident. The third and most common type of stuttering is illustrated by the speech of those persons in whom there are tension and spastic-like contractions, primarily of the tongue but also of other muscles of speech, such as the soft palate, the vocal cords and the lips. The mus-

cles employed in the formation of words start to make the sound and then, instead of relaxing so that the succeeding phonation can occur, become so tense as to interrupt the formation of the word. The effort of the subject to force the word through results in further tension of the tongue and other speech organs, so that the stuttering sounds are increased. The fourth type of stuttering is more the result of a temporary and "emotional" aphasia than a true stuttering. Patients in this group begin to speak and then suddenly forget the word which they wish to use. The word "is on the tip of the tongue," but like the aphasic patient who knows what the object is but cannot name it, even though he recognizes when the object is incorrectly named, so this type of stutterer cannot recall the word he wishes to use. A substitute word will frequently come to the fore and may be uttered simply and perfectly.

These several types tend to merge into one another, so that one frequently sees a patient holding his breath, making many facial grimaces, not being able to think of the word he wishes to use and then bringing forth a battered and unrecognizable form of a word. In practically every patient there tends to be marked general tension, which the patient too often insists is the result of his self consciousness, rather than the causal factor of the speech symptom. The tension of the speech muscles first originates from general tension, and, although by the stuttering a vicious cycle is started in such a manner as to increase self consciousness, the element of primary importance to treat is not the speech, but the tension behind the speech.

ANALYSIS OF CAUSES

In practice it is extremely difficult to distinguish between those symptoms of the ear, nose or throat which are without lesion or associated with minor organic lesion and those which are entirely on an organic basis. Ringing in the ears, pain in the ears, headache, pain in the throat and other symptoms which are usually considered to be on a structural basis can also be on a "functional" basis.[1] Moreover, many of the patients with these symptoms have actual organic disease, on which have been superimposed psychogenic factors, so that diagnosis and treatment become doubly difficult.

Certainly the diagnosis of psychoneurosis should be made only after a thorough physical examination has shown an absence or insufficiency

1. Such a differentiation between structure and function indicates merely preponderance of one without exclusion of the other. Any living structure has function, and any function implies some primary structure. Thus the one and same functional change may result from causes either primarily (not exclusively) organogenic or psychogenic (functional). The heart rate may be greatly increased by such an organic compound as thyroxin or by such an individual, variable functional cause as witnessing a prize fight.

of organic findings. In addition, a definite series of procedures should be followed in eliciting the presence of complications and the causes of the emotional unrest behind the neurotic symptoms. Too often the physician makes up his mind early as to the type of condition present and, as a consequence, overlooks factors which would establish an organic basis for the ailment. In pursuing a systematic procedure one should (1) determine in minute detail the nature of the complaints as to the ear, nose and throat, (2) determine the presence of symptoms in the rest of the organism, (3) obtain a detailed history not only of the physical symptoms but of those psychogenic and situational forces which have produced emotional difficulties, (4) make a thorough physical and laboratory examination and (5) evaluate and crystallize in one's own mind the relative roles played by the various causative factors and evolve a plan of therapy which can be formulated to the patient.

By following such a procedure, the physician may discover that the exact description of the complaints will reveal symptoms which are atypical and which occur in such a fashion as to cast doubt on the presence of any physical lesion. For example, a patient complained of a pain in the nose; in giving a further description he said, "The pain started in the nose and traveled down the body, just as if a thin wire ran from the nose down the body and out the rectum." Such a weird description of pain, not found in the original complaint, immediately identified the patient as suffering from some psychopathic illness, which turned out to be schizophrenia. Many valuable clues may be obtained by determining in detail the "what," "when" and "where" of each symptom. In the second place, the specialist will do well to leave his special field and inquire into symptoms present in the entire organism, for such inquiry will often reveal complaints which are almost obvious in their suggestion of general tension or symbolism. The presence of neurotic symptoms elsewhere in the body will indicate an added need for caution in evaluating the complaint in the ear, nose or throat. Third, the detailed history of the onset of the complaints and the association of these complaints with coincidental emotional upheavals will furnish clues to the nature of the illness. It is always advisable to inquire as to the emotional status of the patient, particularly shortly before the onset of the symptoms.

Thus Mrs. D. complained of total deafness, which was shown later to be hysterical in nature. The deafness came on suddenly one day in March, and no physical cause could be determined. Investigation of the emotional status of the patient at the time of onset revealed that her husband had died three months before and she had had to live with her two children at the home of her relatives by marriage. The mother-in-law, a highly neurotic person, continuously blamed the patient for her son's death, and this blame superimposed on her own sorrow, in addition to the financial dependency, was sufficient cause to bring about a neurosis characterized by the symbolic deafness (the wish not to hear).

Such investigations of emotional background are essential in the analysis of causes of the tension or symbolism. Of course, no diagnosis should be made until, after this history taking, a thorough physical and laboratory examination is made.

One youngster of 12, for example, who complained of severe headaches, was called "a neurotic, overly attached to his mother." A physical examination and the usual otorhinolaryngologic examinations gave negative results. Psychotherapy was instituted, but, though it enabled the patient to bear the pain, there was no relief from the headache. After some months, a roentgen examination of the sphenoid sinus was ordered. A cloudy appearance of the sinus was revealed, and the moment drainage was instituted, the headache disappeared.

Such experiences occur with sufficient frequency to make it important that the physician exercise every precaution before making the diagnosis of "neurotic."

On the other hand, the physician should not swing to the opposite extreme and refuse to make a diagnosis of neurosis when all the evidence points in that direction. He should emerge from his final summing up and evaluation with a definite comprehension of (1) how much organic disease is known to exist, (2) how large a role is played by psychopathic conditions, (3) what procedures are necessary for further diagnosis and (4) what plan of treatment should be followed to correct the physiologic and psychologic disease that is known to exist. In the final analysis the diagnosis and the treatment of the patient depend not on mechanical tests but on the judgment of the physician. Too often the physician postpones his judgment in favor of a laboratory report. It is important that he be clear in his own mind as to what he does know and what he needs to know.

TREATMENT

Treating a symptom while neglecting its cause or treating a part without paying attention to the total organism is poor medicine. There is no local medication, surgical procedure or physical therapeutic measure which can really cure a neurotic complaint of the ear, nose or throat when the cause of the symptom is tension present in the total organism. The treatment of the local pain in the ear or of the aphonia, to be permanently effective, needs to be directed toward the tension which has produced these symptoms, and the physician needs to consider the entire person as he thinks, feels and lives in relation to himself and to his environment.

While such general treatment is usually time-consuming and generally removed from the interest of the general practitioner, there is much which can be done even in relatively short visits.

In the first place, the physician should avoid stating that the patient's complaint or symptom is without organic foundation. For, even if the symptom is a psychoneurotic one, there usually is some

change in the vasomotor system or in the secretion from the mucous membrane or in the contraction of the muscles or in the irritability of the nerve fibers—a change which is at the basis of the symptom. In other words, the psychoneurotic symptom is a symptom of focal physiologic tension and there is some focal lesion. The fact that the focal lesion has resulted from disturbed activity of the autonomic nervous system, which disturbance in turn was caused by some general emotional difficulty, does not alter the fact that there is some actual point on which the patient can focus and on which there is some reason for focusing. Consequently, if the physician tells the patient that there is no reason whatsoever for the pain or symptom the patient will object strenuously because of his actual sensations. The patient feels the pain or other symptom, and neither he nor the physician can eliminate it by mere verbal denial.

Instead of denying the existence of the symptom, the physician should explain to the patient just what his sensation is, how it has come about and what physical and emotional elements enter into its formation. When the patient has a logical explanation of his ailment, he can be turned into an ally in combating the illness, instead of a foe who is in constant doubt of the physician's ability. Such explanations need to be governed by two modifications: (1) the level of intelligence of the patient determines the amount of information given, and (2) the explanation is diagrammatic and not necessarily scientifically accurate in form, so that the patient can grasp easily the principle of the production of the symptom. There is great advantage in the giving of such an explanation, for not only can the patient use the knowledge gained in the correction of his illness, but, what is often more important, he is thereby partially immunized against the persuasive and all-promising claims of charlatans.

In the presentation of the formulation of the illness it is important to be frank and factual. The patient's cooperation is greatly increased when he sees that the physician is sincere both in his diagnosis and in his attempt to explain the illness to the patient. However, using frankness without tact is like drowning a man dying of thirst. The truth, like water, is essential, but both need to be presented at the proper temperature and in the right doses. The technic of this presentation consists of stating to the patient: (1) that one can understand that he is suffering a great deal from the ailment and can sympathize with him; (2) that, whereas there is some pathologic condition at the site of the complaint, the condition itself is the result of malfunction of the integrating systems of the body and is intensified by the emotional difficulties under which the patient is laboring; (3) that the local pathologic condition resulting from focal tension can be rather quickly cleared up if the patient's concerns and anxieties are properly dealt with.

Local treatment should rarely be given to the ear, nose or throat and should consist in that medication which is mildly antispasmodic, which is sedative or which improves the circulation. General hygienic measures should always be instituted; for example, the importance of proper diet, sleep and exercise should be emphasized. It is important to remember, however, that in giving local treatment or any therapy directed at the minor pathologic condition, it is of utmost importance to emphasize to the patient that such local therapy is partial and that the cure lies primarily in the patient's doing all that is possible in clearing up the basic emotional difficulties.

In dealing with the emotional problems of the patient the average physician does not need to utilize special technic, such as free association and analysis of dreams. If the physician will employ a few common sense rules of mental hygiene and will discuss the patient's problems in a detailed and specific manner, a great deal of value will be accomplished. The first rule of mental hygiene is to bring out into the open all the problems which disturb the patient. Second, these problems need to be faced objectively and factually. The patient must learn to deal with a personal problem as if it were a complex algebraic problem capable of solution; i. e., he must obtain a solution only by pure reason and without emotion. Third, the patient needs to set down, again in the most objective of manners, the possible solutions to the difficulties under which he is laboring and to decide which possibility he can work through most successfully. This reasoning must be done without permitting emotional influences and apprehensions of danger to force a false selection of plans. Fourth, each person has to learn (and a person can learn if only he will persist in his efforts) to face the unpleasant solutions without becoming emotional or feeling persecuted. He needs to learn to accept without undue fear, without excessive digust and with the necessary courage any of the unpleasant situations which arise. Fifth, the patient, having decided what to do and having organized his attitude so that there are courage and resolve to carry the plan through, must then put the problem out of mind. To be able to forget about one's problems depends on one's training, and the more the person has been "a chronic worrier," the more difficult will it be for him to dismiss his problems. However, it has been demonstrated repeatedly that most persons can learn to put aside their difficulties if they follow the procedure listed here and if they persist in retraining themselves. Sixth, the patient needs to learn to provide outlets for his energy by means of work, recreation and social activity.

These principles of mental hygiene not only must be intellectually understood by the patient but must become part of his automatic reaction pattern. These mental hygiene responses need to become automatic, in order that the organism shall not respond immediately to any irritant with tension and consequent symptoms which need correc-

tion. And automaticity of response, healthy or unhealthy, is brought about only by training, by repeated and persistent efforts. Consequently, when the patient returns uncured after the first visit, the indication is not that the physician's prescription is at fault but merely that it has not been taken often enough to have an effect. At the subsequent visits the physician needs to stress more and more the need for learning the management of those emotional disturbances which are at the bottom of the symptoms of tension or symbolism.

The preceding statement merely indicates, in outline form, the steps which the average physician, not trained in psychiatry, can take in dealing with neuroses of the ear, nose or throat, or for that matter, of any portion of the human anatomy. Practice will enable the physician to distinguish the important elements in the illness of any given patient. When, however, the patient's otorhinolaryngologic symptoms are part of a general and severe neurosis, the problem is of special psychiatric interest, and further methods are required in readjustment of the personality.

Finally, one may use the methods of suggestion and of hypnosis in relieving a neurotic condition. When these methods are used, however, it must be remembered that the physician has not changed any of the causative mechanisms. Simply suggesting away the symptom while the patient is under hypnosis is overlooking the unhygienic mental state which is at the bottom of the illness. This overlooking of the cause is the reason why most "cures" by hypnosis are of short duration.

SUMMARY

Psychoneurotic symptoms fall into two categories, symptoms of tension and symptoms of symbolism. Symptoms of tension may be general, such as irritability, insomnia and "worrying," or focal wherein the involvement primarily of the autonomic nervous system gives rise to tachycardia, ringing in the ears, rhinorrhea, mucous colitis and other ailments. Symbolic symptoms are those in which the symptom represents, unconsciously to the patient, certain underlying emotional disturbances. The ear, nose and throat are frequently involved in the states of tension and symbolic states, and the treatment of the neuroses of the ear, nose and throat must follow the treatment of the underlying emotional conditions. Such treatment follows the procedure of analyzing the nature of the underlying causes, outlining to the patient the mechanism of his symptom formation, demonstrating to him how he should deal with the problems that confront him and instilling principles of mental hygiene. The use of suggestion, hypnosis and drugs should be restricted as adjuvant therapy, for these technics do not aim at removing the cause of the neurosis.

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SYMPTOMS OF GASTROINTESTINAL ORIGIN IN THE EAR, NOSE AND THROAT

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Every practitioner of otorhinolaryngology is from time to time confronted by cases in which the causation is so obscure that diagnosis presents seemingly insurmountable difficulty. When all organic causes have been eliminated and treatment of the organs involved has not given satisfactory results, the possibility of allergic complications should always be considered. Particularly should the clinician confronted with irritability or actual disturbance of function in the ear, nose or throat consider the possibility of gastrointestinal disturbance as the chief etiologic factor.

Interference with the physiologic function of the gastrointestinal tract frequently sets up a toxic condition in the lower part of the bowel which causes it to become a reservoir for infection, later to be conveyed by the lymphatic and vascular systems to distant parts of the body. An intimate relation exists between the gastrointestinal tract and the ear, nose and throat, which makes such infection peculiarly apt to find an outlet in these structures.

Though these facts are well known, there is a surprising dearth of literature on the subject. It is my aim in this paper to direct attention to the frequency with which gastrointestinal allergy is manifested in the ear, nose and throat and from my own experience to draw certain deductions, which I trust will be of service to my fellow practitioners.

Chief among the complaints for which no pathologic condition in the ear, nose or throat exists as a causative factor are:

1. A persistent postnasal drip, after disease of the sinuses has been definitely ruled out by thorough examination of these cavities.

2. Vertigo and a sense of fulness in the ears when a pathologic condition in the aural region which will account for such symptoms cannot be found.

3. Rhinorrhea, associated with sneezing, recurring colds, cough and other manifestations of hay fever.

4. Dryness and stuffiness of the nasal mucosa, accompanied by headache, "burning" of the eyes and a sensation of pressure at the root of the nose.

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5. Ocular palsy involving the extraocular muscles, accompanying frontal headache.

6. The "lower half headache" of Sluder, consisting of pain about the eye, the upper jaw and the teeth, extending to the zygoma and temple, with earache and pain in the mastoid, extending thence to the occiput, neck, shoulder, scapula, arm, forearm, hand and fingers.

When the most careful survey has failed to reveal any organic lesion to account for the conditions just enumerated, the inference that the causal factor is located in the gastrointestinal tract is fairly strong. The primary site of intestinal toxemia is most often located in the colon, but whether the conditions mentioned result directly from this focus or are produced secondarily by hepatic disturbance, which, in turn, has been inaugurated by the liver's absorption of toxic products from the intestine, I have not yet been able to find out.

Though it is generally recognized that gastrointestinal allergy substantially contributes to the conditions in the ear, nose and throat of which many of my patients complain, I feel that this underlying cause has not been sufficiently emphasized, either in literature or in clinical practice. The toxemia on which these states are dependent may be caused by spasticity or irritability of the colon or by mild forms of chronic ulcerative colitis, which often go unrecognized. The gastric secretion may be quite normal, but the presence of indican in the urine gives evidence of the existence of intestinal toxemia, and if roentgen examination is carried out after ingestion of a barium sulfate meal, the cecum will often be found to be markedly dilated, and a local or general atony, with resulting intestinal stasis and retention of waste products, will be in evidence.

Factors which, singly or collectively, may act to produce in the ear, nose and throat the conditions under discussion may be grouped as follows:

1. Bacterial invasion.

2. Disturbances in the motility of the bowels.

3. Disturbances in the innervation of the bowels or endocrine imbalance.

4. Alterations of the normal intestinal secretions, resulting in various lesions of the intestinal mucosa.

Establishment of the existence of one or a combination of several of these conditions will at once offer an explanation of otherwise obscure pathologic states in the domain of otorhinolaryngology.

Investigating multiple sensitizations in allergy, William Lintz [1] found in a thorough study of 500 patients that 472 were suffering from gastro-

1. Lintz, W.: Gastrointestinal Allergy, Rev. Gastroenterol. **6**:320-332 (July-Aug.) 1939.

intestinal forms. Incompletely digested foreign proteins gain access to the circulation not alone in persons who are sensitized but in presumably normal persons as well. In the sensitized person, however, the entrance of the offending protein provides an agent exciting to the cells of the gastrointestinal tract, which thereupon react in an abnormal manner. "The severity of the symptoms," said Lintz, "is determined by the sensitivity of the patient and the quantity of the allergen." He expressed the belief that these sensitized patients have a congenital deficiency of a ferment or internal secretion which normally acts to neutralize the toxic effects of the protein. ". . . the only difference between normal and allergic people is one of degree. They, the normal, can tolerate larger doses of the allergens." Though it is estimated that no more than 10 per cent of the general population suffers from allergy of major importance, it is highly probable that the great majority, at one time or another, produce mild and irregular allergic symptoms, that go unrecognized as such. How often does one hear that a certain person "can't eat" this or that because it "makes him bilious," "gives him headache" or causes him to "break out all over"? By avoidance of the offending protein he "cures" himself without recourse to the physician. Lintz found that gastrointestinal allergy was by far the most prevalent form of this reaction.

When a patient presents himself complaining of trouble with the ears, nose or throat, the cause of which is not immediately apparent on physical examination, a most thorough family and personal history should be elicited without delay. The personal history will nearly always show that the patient has previously been subject to "attacks" which can be attributed to certain foods, while he will often present other allergic manifestations besides those referable to ear, nose or throat. From at least 70 per cent of these patients a family history suggestive of allergy or giving positive indications of it will be forthcoming. The majority, especially among the female patients, will present a history of one or many operations undertaken to correct some supposed lesion of the digestive tract, the results of which have been unsatisfactory. Lintz remarked that "nose and throat operations are so frequent that they should be considered a symptom of the disease."

Of course, organic disease of the intestinal tract must be ruled out before one attempts to establish gastrointestinal allergy as the causative factor in otolaryngorhinologic manifestations. The usual dermal tests for allergy are of less value with these patients than are "trial diets." The autonomic nervous system plays a most important part in the production of gastrointestinal allergy. This explains why a person may tolerate a certain food at one time and suffer an allergic reaction from

no greater quantity of the same food at another time. Conditions prevail which make for overstimulation of the vagus; at another time these conditions may be entirely lacking. At times the attack may be induced by a food which forms no regular part of the patient's dietary. He will relate that this "strange" food "disagreed with" him. Again, the offending protein may be present in some common article of diet, and in these cases the regular diet will usually, on investigation, turn out to be deficient in vitamins.

I consider these investigations into the patient's dietary habits to be of extreme importance, for it has been my experience that many patients fail to be relieved of their symptoms by medication or operations directed toward cure of the local manifestations because of the underlying gastrointestinal allergy, which nullifies every attempt at local alleviation. Under the ministration of a competent gastroenterologist many of the otorhinolaryngologist's "failures" will become brilliant "successes." The physician who discovers the gastrointestinal condition will often be hindered by the patient's unwillingness to have suitable roentgenographic investigations instituted, because of the expensive involved. This places the conscientious practitioner in an awkward position, but under the circumstances his only recourse will have to be careful regulation of diet and the correction of any local conditions which tend to aggravate the underlying allergy.

The patient suffering from gastrointestinal allergy is usually recognized without much trouble—almost at sight. He is one who takes little outdoor exercise, is impatient of any dietary restriction and is generally lacking in physical energy. Constipation is a frequent complaint, and most patients speak of trouble with intestinal "gas." But even if the patient insists that his bowels move regularly, the examiner should not permit himself to be led away from his conviction that a thorough examination, with tests for allergy, is essential.

REPORT OF CASES

CASE 1.—Mrs. J. L., a resident of South America, 52 years of age, had had for three months a persistent headache in the left lateral region. Though constantly under a physician's care, she had had no respite from the pain except that obtained by the use of analgesics. Inspection and roentgenographic examination of the teeth, nose and accessory sinuses revealed nothing abnormal, but questioning brought out that a week or more before the onset of the persistent headache the patient had suffered a mild "intestinal upset" with diarrhea, which she had attributed to some fish eaten the previous day. There were no complaints referable to the intestinal tract at the time of this examination, however, and the patient insisted that except for the headache she felt perfectly well.

This history, together with the failure to find any organic lesion to account for the headache, suggested a septic condition and dysfunction of the intestinal tract as

the underlying factor. Regulation of diet and the administration of intestinal antiseptics shortly proved the correctness of this assumption. The headache soon ceased, and after six weeks the patient reported that it had not returned.

Comment.—This case illustrates the importance of making a thorough examination and eliciting a complete history, not confined to the particular organs with which the examiner is especially concerned. Whenever there is lack or absence of local evidence in the ear, nose and throat, it is essential to make a search for causal factors in other parts of the body.

CASE 2.—Mrs. M., aged 33 years, the wife of a physician, was brought for examination. For seven months she had been suffering from multiple arthritis, which involved the arms, wrists, knees, ankles and feet and caused such severe suffering that opiates had had to be used for its control. For four and a half months she had been subject to attacks of vertigo. The use of opiates induced constipation and intestinal gas, which had been temporarily relieved by the insertion of a rectal tube and the administration of high enemas. The attacks of vertigo first appeared when the patient's condition improved sufficiently to permit her being propped up in bed and later occurred whenever the position of the head was changed suddenly. With subsidence of the pain, smaller amounts of opiates were required and the condition of the intestinal tract improved, but elimination remained inadequate, and, even when the patient was able to be up and around, the vertigo continued to be annoying.

Two otorhinolaryngologists, in consultation with a neurosurgeon, had made a diagnosis of tumor of the eighth cranial nerve and had advised operation. It was at this time that I was consulted. I could not concur in the opinions of the other physicians, because the early otologic symptoms of acoustic nerve tumor were not present. There was no tinnitus and only a slight deafness and no paralysis of the equilibratory apparatus. My diagnosis was toxic labyrinthitis, secondary to absorption of toxic substances from the intestinal tract. The patient was referred to a gastroenterologist; a treatment was outlined which gradually freed the patient from the vertigo, so that eight months later it had entirely disappeared.

Comment.—In this case one can note the effect of the constant administration of opiates on the gastrointestinal function. Intestinal stasis was inaugurated, which, in turn, was contributory in setting up the toxic condition in the labyrinth.

CASE 3.—Two weeks after Mrs. S. C. P. arrived from Germany double vision (ocular palsy), accompanied by a dull frontal headache, developed. I was summoned by the family physician to the patient's home; he informed me that he feared the patient had encephalitis lethargica and desired me to make a thorough exploration of the sinuses. This I did, but the results were completely negative. I then suggested an investigation of the gastrointestinal tract, and in the course of this it was brought out that while crossing the Atlantic, about ten days before, the patient had had a mild "stomach upset," with nausea and vomiting, followed by slight diarrhea. The family physician would not admit that there could be any connection between this illness and the present ocular condition and demanded another sinal examination. When the results were again negative, at my request

another otorhinolaryngologist was called in, but his report was likewise completely negative. Further consultation with an ophthalmologist and a neurologist resulted in a return to the tentative diagnosis of beginning encephalitis lethargica, and a grave prognosis was given the family. The husband, greatly alarmed, that evening consulted me privately and expressed his desire that his wife be examined by a competent gastroenterologist, who had previously cured him of ulcer of the stomach. Under this physician's direction colonic irrigations were begun, with the administration of intestinal antiseptics. Within three weeks the diplopia and headaches had had disappeared, and the patient made a satisfactory recovery.

CASE 4.—H. M. B., aged 38 years, presented himself with a history of stuffiness of the nose and a drip from the back of the nose into the throat, of many months' duration. Examination showed nothing more than a red and swollen condition of the mucous membrane of the middle and inferior turbinates; postrhinoscopic examination revealed a mild congestion of the lining of the nasopharynx, redness and swelling of the posterior tips of the inferior turbinates and a few strings of thin mucoid material clinging to the lateral walls of the epipharynx.

To rule out sinal disease the following technic was employed: The nose was sprayed with 0.5 per cent solution of ephedrine sulfate in physiologic solution of sodium chloride. To obtain shrinkage farther back, the spray was repeated after five minutes. A cotton applicator saturated with the 1 per cent solution of ephedrine sulfate was brought into service to shrink the membranes surrounding the openings to the sinuses. The patient was placed face down on the table, with head rotated to one side as it hung from the table's edge. After this position was held for three minutes, the head was rotated to the opposite side. Thereafter the erect position was assumed, and a careful posterior and anterior rhinoscopic examination was made. After this inspection, mild mass-suction was applied to the nose, and the anterior and posterior rhinoscopic examination was repeated.

This examination revealed no evidence of sinal disease. Stereoscopic roentgenograms of the sinuses showed that they were normal.

Treatment.—Dietary treatment was undertaken. A lower intake of protein was advised. Fresh and cooked fruits were to be taken freely, while an alkali was prescribed to reduce gastric acidity. To combat a possible low grade intestinal infection, soricin (sodium ricinoleate), 10 grains (0.65 Gm.) three times daily, was likewise prescribed.

Further Course.—Within a week the patient reported that the postnasal drip had appreciably diminished and the stuffiness of the nose had somewhat lessened. Seen two weeks thereafter, the patient stated that his condition was much improved. One month after treatment was instituted, reexamination of the nose and throat showed much less congestion, and the accumulations of mucoid material in the epipharynx had disappeared.

CASE 5.—A chauffeur, 41 years of age, complained of unilateral pain, neuralgic in character, involving the right eye and the parietal and postauricular regions and extending down to the shoulder, arm and forearm. The first attack had occurred fourteen months previously, since which time attacks had taken place every two to three weeks, at times lasting for several days.

On examination the nose, sinuses, throat and ears appeared normal, and stereoscopic roentgenograms of the sinuses showed them to be clear. I noted, however, that the patient's skin had a "pasty" look, like that of one who is physically below par. Questioned as to his eating habits, he admitted that he took

his meals hurriedly, consuming as many as three cups of coffee with each meal. Often when he had not time for a full meal he would gulp down coffee and dessert only, usually a piece of pie or cake. He denied that he had constipation but admitted the presence of considerable intestinal gas.

The advice given was to take more time for meals and, if possible, to omit fluids with meals. The patient was to drink water freely between meals and to take less coffee. As an intestinal antiseptic, dihydranol (2,4-dihydroxyphenyl-n-heptane), two capsules (5 grains [0.3 Gm.]) immediately after each meal, was prescribed. A week later the dose was increased to three capsules. In three weeks this patient reported that he had suffered one mild attack of neuralgic pain, but that it had extended no farther than the postauricular region and had lasted no more than two hours. Two months later he reported that he had had no further painful attacks and that his general health had vastly improved.

COMMENT

In the majority of these gastrointestinal conditions, the aim of treatment is to restore the colon's normal tone and to overcome any existing toxemia. This can be accomplished by an anticonstipation diet in which fruits and vegetables predominate, but in which there is not too much roughage, especially if a spastic condition is present. Drastic purgation must be strictly avoided, but retention enemas of liquid petrolatum U. S. P. and the ingestion of adequate quantities of this oil, with or without agar, will help to overcome constipation.

I have found intestinal antiseptics valuable. My patients have obtained benefit from the administration of soricin (sodium ricinoleate) and also dihydranol (2,4-dihydroxyphenyl-n-heptane). Soricin is given in capsules—each containing 10 grains (0.65 Gm.) of the drug in a bland oil excipient. One capsule is given three times daily. Dihydranol is also put up in capsules—2½ grains (0.15 Gm.) in 8 minims (0.49 cc.) of olive oil. The dosage is 2 capsules immediately after each meal, increased to 3 capsules if necessary. These drugs are alternatives, soricin being the choice in milder cases, while dihydranol, which is somewhat of a stomach irritant, with greater antiseptic properties, is reserved for the treatment of more obstinate conditions. Treatment with either should be continued from three to six weeks, depending on the amount of improvement in the patient's general condition.

The use of a combination of metaphen and agar which I formerly prescribed extensively has largely been discontinued because it was bulky and less convenient for the patients to use. It remains useful, however, and occasionally offers some advantages, in that the patient may prepare it for himself. Four heaping teaspoons of agar and 4 teaspoons of metaphen (1:500) are added to one quart of water, and the mixture is boiled until the agar and metaphen have entirely dissolved; a flavoring of raspberry syrup, according to taste, is added.

The mixture is placed in the refrigerator and allowed to congeal. The dosage is 2 heaping tablespoons night and morning.

In addition to the dietary and medicinal treatment outlined, there is instruction as to general hygiene. The patient should be impressed with the importance of adequate rest, moderation in the use of coffee and alcoholic beverages and the need of outdoor exercise suited to his age and previous habits. These simple measures have frequently proved far more effective than surgical intervention and should always be resorted to in any case in which there is no significant pathologic condition of the ear, nose or throat to account for the symptoms presented. Even if they fail, no harm will have been done, and the patient will undoubtedly be in better condition to withstand operation, should it later prove necessary.

SODIUM MORRHUATE THERAPY FOR CHRONIC SIMPLE, VASOMOTOR AND HYPERTROPHIC FORMS OF RHINITIS

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Chronic rhinitis with engorgement or congestion of the turbinates is a common disorder. Pathologically and clinically it has been classified as turgescent, intumescent, vasomotor, hyperesthetic, allergic, hypertrophic and hyperplastic rhinitis. The variations in nomenclature are somewhat confusing.

These types of rhinitis cause nasal obstruction. The chronic simple form is characterized by chronic inflammation and swelling of the soft tissues with distention of the blood sinuses of the turbinates and with a varying degree of proliferation of connective tissue, from prolonged or frequent recurrences of acute inflammation, chronic sinal disease, anatomic defects and related disorders. The mucous membrane appears swollen, smooth and reddened. Patients with rhinitis of the intumescent form complain of nasal obstruction, particularly night and morning, with frequent headaches of varying intensity. Hypertrophic rhinitis can be considered as a later form, differing from the chronic simple form by its association with an enlargement of the cellular elements of the turbinates, with some increase in the fibroblastic tissue, thickening, and permanent dilatation and increase in the number of blood sinuses and glandular elements (serous and goblet cells). Since hypertrophy actually means an increase in the size of the cells that are present, the use of the term hypertrophic is somewhat misleading, for there is some hyperplasia, or increase in the number of cellular elements. As this form progresses, it eventually produces a true hyperplasia and frequently a granular lobulated structure on the posterior portion of the turbinates, spoken of as a "mulberry mass." The vasomotor type of rhinitis is also referred to as hyperesthetic rhinitis, rhinorrhea, turgescent rhinorrhea, nervous coryza, summer catarrh or perennial hay fever. It often simulates mild allergic rhinitis. In fact, as more is learned from the point of view of allergy, it is becoming apparent that many of the instances of rhinitis belong in this category. The usual symptoms are like those

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of seasonal hay fever, but they are milder and persist with exacerbations and remissions, frequently with sensations more or less of a constant "head cold." There may or may not be paroxysmal sneezing, particularly in the morning, with a watery or mucoid discharge. Pathologically, the mucous membrane is edematous and pale, and frequently there are polypoid formations. There are usually a desquamation of cilia and an increase in goblet cells. The tunica propria is swollen, and there is an infiltration with plasma cells, fibroblasts and some eosinophils. The seasonal hay fever, or allergic rhinitis, presents a similar but more pronounced pathologic picture. The symptoms are also more acute, with paroxysmal sneezing, watery nasal discharge with increased eosinophilia, marked nasal obstruction, with its associated discomfort, lacrimation and inflammation of the ocular conjunctiva.

As with other diseases or disorders for which most therapeutic measures are inadequate, many kinds of treatment, mostly empiric, are administered. The ideal method of treatment should include the removal of the causative factors. Anatomic defects of the nose should be corrected, diseased sinuses and other foci of infection should receive proper treatment and any allergic factors should be annihilated or treated so far as it is feasible. However, in many patients, rhinitis persists in spite of all therapeutic and surgical measures. In fact, the tissue reactions are aggravated after prolonged energetic use of various astringents and topical applications.

In an effort to give these patients relief from the disagreeable effect of nasal blockage, headache or feeling of fulness in the head and other associated manifestations of nasal congestion, physicians have made use of the following procedures:

1. Application of chemical caustics or escharotics to the surface mucosa—(*a*) phenol, (*b*) trichloroacetic acid, (*c*) resorcinol, (*d*) chromic acid.

2. Physical therapy—(*a*) treatment with the galvanocautery or medical diathermy, (*b*) ionization, (*c*) application of radium or roentgen ray.

3. Topical application of astringents—(*a*) ephedrine, (*b*) epinephrine, (*c*) amphetamine, (*d*) propadrine hydrochloride, (*e*) neosynephrin hydrochloride.

4. Internal medication (oral or parenteral) with (*a*) potassium iodide, (*b*) free iodine, (*c*) vaccines, (*d*) histamine and histamine-like products, (*e*) desensitizing agents, (*f*) benzedrine.

5. Administration of sclerosing preparations by submucous injection —(*a*) alcohol, (*b*) glycerin, (*c*) quinine and urea hydrochloride, (*d*) quinine lactate, (*e*) phenol, (*f*) dextrose, (*g*) sodium morrhuate.

Concentrated phenol was applied to the nasal mucosa by Palmer.[1] There were sneezing and hypersecretion for a few hours and clogging and congestion for twenty-four to forty-eight hours after the application. Of his 30 patients, 80 per cent showed definite improvement; 12 were free from symptoms from one to nine months. Examination of tissues revealed less glandular tissue in the submucosa and, in some cases, degeneration of epithelium; the blood sinuses had collapsed and the endothelial lining had degenerated in many instances. Chandler[2] obtained excellent results for one to two years. Spivacke[3] treated 16 patients, 10 of whom obtained complete and almost immediate relief and 3 of whom were moderately relieved. There was no scarring of these subjects. Reports on patients treated by streaking the turbinates with chromic acid or trichloroacetic acid compare favorably with those on patients treated with phenol. Disadvantages of using the caustics and the escharotics are the discomfort, the sloughing and the formation of crusts and adhesions. Lewy[4] used a watery paste of resorcinol on the mucous membrane, with favorable results lasting from a few weeks to several months.

Ionization has been enthusiastically supported by some clinicians. Different authors have reported results varying from no benefit to 100 per cent cures. Hollender and Cottle[5] and Demetriades[6] reported good results in the treatment of simple rhinitis and vasomotor disturbances in 1926 and 1927. All but 1 of the 40 patients treated successfully by Warwick[7] were entirely relieved of their hay fever and associated allergic symptoms for more than a year. Nineteen of these patients obtained complete relief for over three years. Warwick did not state how many patients failed to respond to the ionization. He asserted that the success of this treatment depends on the use of the proper machine and electrolyte and that the nose must be properly packed. According

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2. Chandler, G. F.: Hay Fever Cured by Applications of Carbolic Acid to the Nasal Mucosa, M. J. & Rec. **136**:337 (Oct.) 1932.

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5. Hollender, A. R., and Cottle, M. H.: Recent Advances in Treatment of Nasal Accessory Sinus Disease, Eye, Ear, Nose & Throat Monthly **5**:575, 1926.

6. Demetriades, T. D.: Zur Behandlung der vasomotorischen Störungen der Nase durch Iontophorese (Rhino-Kataphorese), Monatschr. f. Ohrenh. **61**:524 (May-June) 1927.

7. Warwick, H.: The Treatment of Hay Fever and Allied Conditions by Ionization, Laryngoscope **44**:173 (March) 1934.

to Hollender,[8] ionization is only occasionally effective in the treatment of hay fever. In cases of vasomotor rhinitis it is beneficial as a prolonged palliative treatment. The lack of unity in results reported by these and other authors, including Sputh,[9] Franklin,[10] Hurd,[11] Tobey [12] and Bernheimer,[13] leaves one confused as to the value of this form of treatment. According to Smit [14] and Dean and his associates,[15] the microscopic results of ionization were: (1) fibrosis and thinning of the tunica propria, (2) thickening and increase in density of the basement membrane, (3) hyperplasia of epithelium and increase in metaplasia, (4) the presence of few goblet cells and (5) great destruction of secretory glands.

Aikens [16] and Scott-Brown [17] obtained good results by the use of radium. Bernheimer [18] reported satisfactory clinical results from irradiation of the allergic nasal mucosa, with no evidence of atrophy three years later. However, the results were negative when the treatment was used for typical seasonal rhinitis. Glas [19] used cauterization effectively in the treatment of hay fever. Hitschler,[20] Wahrer [21] and Fletcher and Friedberg [22] presented evidence of satisfactory results from medical

8. Hollender, A. R.: Ionization as Prolonged Palliative in Vasomotor Rhinitis, Arch. Otolaryng. **21**:448 (April) 1935.

9. Sputh, C. B.: Zinc Ionization in Treatment of Rhinitis and Nasal Accessory Sinusitis, J. Indiana M. A. **24**:461 (Sept.) 1931.

10. Franklin, P.: Treatment of Hay Fever by Intranasal Zinc Ionization, Brit. M. J. **1**:1115 (June 27) 1931.

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22. Fletcher, I. E., and Friedberg, S. A.: Treatment of Hypertrophic and Intumescent Rhinitis, Ann. Otol., Rhin. & Laryng. **15**:73 (March) 1906.

diathermy and the use of the galvanocautery. Horn [23] used submucous electrocauterization with good results. Beck [24] treated over 50 patients for intumescent and hypertrophic rhinitis successfully by submucous electrocoagulation. Francis [25] found that all symptoms of hay fever were markedly diminished by touching the sensitive areas in the nose lightly with an electrocautery. Watson [26] stated that not over 50 per cent of the patients are benefited by the cautery or escharotic methods of treatment.

Turbinectomy has been done for the relief of nasal obstruction, but this method has fallen by the wayside for the treatment of the turgescent type of rhinitis because it is too radical, since the normal physiologic function of the nose is markedly disturbed.

Submucosal injections of 0.5 cc. of 60 to 70 per cent alcohol were reported by Hutter [27] in 1924. Metzenbaum [28] with this form of therapy obtained relief from symptoms within seven to ten days. The patients complained of a sharp burning sensation radiating toward the eyes and over the side of the face and to the teeth. The nose was blocked for two or three days, and there was anesthesia for a few days. A partial or a total loss of normal sensations was also produced. He stated that the nose may drip for a considerable period. Leichsenring [29] reported beneficial results from the treatment in patients with allergic diseases. Vail [30] obtained relief for 50 per cent of his patients for six to eight weeks by the injection of 1 or 2 cc. of 40 to 60 per cent alcohol. Stein [31] injected alcoholic solutions into the nerve filaments through the sphenopalatine foramen. The pain from the injection was sometimes prolonged.

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23. Horn, H.: The Treatment of Intumescent Rhinitis by Submucous Method, Ann. Otol., Rhin. & Laryng. **17**:490, 1908.

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25. Francis, C.: A Nasal Treatment of Hay Fever, Practitioner **132**:614 (May) 1934.

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30. Vail, H. H.: Treatment of Hay Fever by Nasal Injections of Alcohol, Arch. Otolaryng. **18**:651 (Nov.) 1933.

31. Stein, O. J.: Intranasal Injection of Alcohol in the Treatment of Hyperesthetic Rhinitis and Nasal Neuroses, Ann. Otol., Rhin. & Laryng. **31**:1129 (Dec.) 1922.

García Ibañez [32] obtained good results from the injection of 0.5 cc. of quinine and urea hydrochloride. From eight to fifteen days were required for the disappearance of congestion and complete sclerosis. Monson [33] treated 17 patients with quinine and urea hydrochloride and ethyl carbamate, with improvement or cure in 70 per cent of the patients. With quinine lactate, the results were somewhat better, and no sloughing or other unfavorable effects occurred. Hutchinson [34] injected phenol submucosally and maintained that this treatment is better than cauterization. Although Monson [33] used 50 per cent dextrose without particularly impressive results, Baer [35] reported favorably on its use.

Sternberg and Sugar,[36] Bishop,[37] Levine [38] and Meyersburg [39] have reported excellent results from subcutaneous injections of a 5 per cent solution of sodium iodide or of a preparation of sodium iodide with a trace of free iodine in the treatment of vasomotor rhinitis. Some patients required as many as ten injections.

Thus, according to the studies reported in the literature, not only is there a great deal of discrepancy in the results obtained by different investigators using the same preparation, but there are definite objections to the use of most of the preparations which have been employed.

The treatment of varicosities with sodium morrhuate [40] has been definitely established as sound. Since there is enlargement of the vascular sinuses or marked dilatation of the cavernous spaces in chronic simple and other forms of rhinitis under discussion, it has been my purpose in this investigation to determine the effect on such conditions of submucosal

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32. García Ibañez, L. M.: El tratamiento conservador en las rinitis hipertroficas, Bol. Asoc. méd. de Puerto Rico **25**:544 (Sept.) 1933.

33. Monson, L. P.: Treatment of Hypertrophied Inferior Turbinate by Use of Sclerosing Solutions, Arch. Otolaryng. **22**:96 (July) 1935.

34. Hutchinson, C. A.: Sclerosing Submucosal Injections of Turbinates, Brit. M. J. **1**:72 (Jan. 9) 1937.

35. Baer, M.: Zur Injektionsbehandlung der Rhinitis chronica hyperplastica, Monatschr. f. Ohrenh. **73**:108 (Feb.) 1939.

36. Sternberg, H., and Sugar, M.: Ueber therapeutische Beeinflussung nasaler Hypersekretionen (Rhinitis vasomotoria) durch kleine Joddosen, Wien. klin. Wchnschr. **39**:416 (April 8) 1926.

37. Bishop, J. R.: The Treatment of Oto-Rhinological Conditions with Free Iodine Administered Subcutaneously, Laryngoscope **43**:411 (May) 1933.

38. Levine, M.: Free Iodine in Treatment of Vasomotor Rhinitis and the Symptom Complex of Sneezing and Nasal Hydrorrhea, Laryngoscope **43**:39 (Jan.) 1933.

39. Meyersburg, H.: Efficiency of Subcutaneous Free Iodine Therapy, M. J. & Rec. **138**:317 (Nov.) 1933.

40. The product referred to is a 5 per cent solution of sodium morrhuate with benzyl alcohol. Each cubic centimeter contains 0.05 Gm. sodium morrhuate and 0.02 Gm. benzyl alcohol in aqueous solution.

injections of this sclerosing agent. Fishof's [41] report of 7 patients successfully treated with sodium morrhuate is the only one found in the literature.

In order to ascertain whether this form of therapy is desirable for a given patient, it is necessary to give attention first to the following steps:

1. Thorough recording of history—particularly as to sinal disease and allergy.
2. Examination of the nose and throat, including transillumination of the sinuses and roentgen examination if indicated.
3. Correction of any anatomic defects of the nose which may be causative factors.
4. The application of vasoconstricting medicaments by tampon or spray, to determine whether or not the turbinates shrink well.
5. Correction of any ocular disorder which may account for headaches.

The indications for treatment with sodium morrhuate are:

1. Chronic nasal obstruction from engorged turbinates which have failed to respond to the usual types of therapy after two months' trial.
2. Enlarged or turgescent turbinates which shrink well in response to the application of astringents.
3. Postnasal drip, chronic pharyngitis, headaches and neuralgic pains in the forehead and the postauricular, aural and antral regions, with intumescence of the turbinates, causing pressure on the lateral wall of the nose or the septum.
4. Intermittent nasal obstruction of long duration, especially when it is associated with changes of temperature and humidity.
5. Chronic sinusitis with associated intumescence of the turbinates.

The contraindications to submucosal injection of sodium morrhuate solution are:

1. Chronic systemic diseases, such as arteriosclerosis, hypertensive or other types of advanced cardiac disease and specific diseases in which proper treatment of the primary cause should improve the nasal congestion.
2. Anatomic defects of the nasal air passages, such as deviated septum, nasal spurs or enlarged or bullous projections of the bony framework of the concha. Such defects should be corrected surgically; then if nasal obstruction persists after a few months, injection of the sclerosing agent may be done.
3. Acute infections of the nose, throat and paranasal sinuses.
4. Hyperplastic rhinitis in which the enlarged turbinates fail to shrink well after the application of astringent preparations.
5. Definite allegy with nasal manifestations, which can be controlled adequately by desensitization or by removal of the allergen.

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41. Fishof, F. E.: The Treatment of Vasomotor Rhinitis and Allied Conditions with Sodium Morrhuate, Arch. Otolaryng. **27**:413 (April) 1938.

TECHNIC FOR SUBMUCOSAL INJECTION

1. Tampons saturated with a 2 per cent or 4 per cent solution of butyn sulfate are applied to the nasal fossa. One tampon is placed above the inferior turbinate, in the middle meatus. A second tampon is placed in the common meatus and floor of the nose. These are left in place for five to ten minutes. Larocaine hydrochloride is the anesthetic of second choice for topical application.
2. A number 22 spinal puncture needle on a tuberculin syringe or other long 1 cc. syringe is satisfactory for injecting the inferior turbinates. A gold needle, which can be curved, can be used advantageously to inject the middle turbinates.
3. It is usually advisable to use only a small amount of sodium morrhuate for the first injection, as it is possible for a patient to have an idiosyncrasy to this as well as to most other drugs. Five-tenths cubic centimeter can be used if the patient tolerates the preparation. Even though this precaution was not taken in most of the cases reported here, no untoward reactions were observed.
4. The tampons are removed, and a small strip of cotton is placed along the floor of the nose to absorb any of the sclerosing agent which may accidentally escape into the nasal passage. The nose may also be sprayed with a bland oil to protect the mucosa in case some of the material should escape.
5. The bevel of the needle is turned toward the septum. The anterior end of the inferior turbinate is entered at the mediosuperior angle and is followed along the bony margin toward the posterior tip. The sodium morrhuate is injected gradually as the needle is slowly withdrawn.
6. A pledget of cotton is placed in the nares to stop bleeding from the needle puncture.
7. After about five minutes the pledget of cotton and the strip of cotton on the floor of the nose are removed.
8. A solution of cocaine, ephedrine or neo-synephrin hydrochloride is used to shrink the nasal mucosa.
9. It is advisable to inject only one side of the nose at a sitting. After one week the other side of the nose may be treated.
10. It is best to allow two weeks to elapse before another injection is made in the same turbinate.
11. Since the middle turbinates are not easily injected, unless a curved needle is used, injections at different points on the medial and lateral surfaces of the dependent border are necessary to obtain satisfactory results.

A 2 per cent solution of butyn sulfate used as a topical anesthetic serves the purpose admirably, if applied by means of tampons extending to the posterior nares. Two patients on whom several applications of cocaine previously had been made required a 4 per cent solution of butyn sulfate. A 2 per cent solution was used in 2 patients in whom toxic manifestations previously had developed when applications of cocaine were made. The butyn sulfate produced excellent anesthesia, and the patients gave no evidence of hypersensitivity to the drug. Larocaine hydrochloride, 10 per cent, can be used instead of butyn

sulfate; even though slight shrinkage does occur, it is not enough to interfere greatly with the injection procedure. Cocaine produces marked shrinking. There are two reasons for not shrinking the nasal mucosa before making the injections:

1. It is much easier to make the injections, and the chance of losing the preparation in the nasal cavity because of mucosal perforation is correspondingly less.
2. The sclerosing agent is given a better chance to disseminate throughout the submucosal tissue.

The distribution of the oil is further augmented by the application of a shrinking solution by spray or tampon soon after the injection is made.

A taste of fish oil and a slight burning sensation are produced if some of the sodium morrhuate solution accidentally escapes into the posterior nares or the nasopharynx. No untoward effects have been observed from this. However, if a patient should complain of these sensations, the nose can be sprayed or irrigated with ephedrine in saline solution or physiologic solution of sodium chloride. The immediate effect of submucosal injections of sodium morrhuate solution on the patients varies. Most patients experience little discomfort. Some have pain in the maxilla, ear, teeth and frontal region on the side injected. It generally lasts but a few minutes, although a dull aching may persist for several hours. This can be relieved with the usual coal tar derivatives. A state of congestion of the turbinates follows the injection, with an excess of mucous secretion. This lasts for one or two days, after which the patient notices a gradual improvement in breathing through the nostrils.

As mentioned previously, the nasal tissue was shrunk after the injection on the theory that the sclerosing agent would be forced into submucosal spaces which otherwise would not be reached. The use of nose drops every two or three hours was instituted and continued for several days after the treatment to give the patient relief from the congestion which occurs and because it was thought that shrinking might aid in keeping the vascular spaces collapsed until the therapeutic effect of the sodium morrhuate was fairly well established. This belief is certainly debatable, since it is impossible to keep tissue constantly contracted with such topical applications.

The study presented in this paper included 50 patients treated by me and 8 patients treated by Dr. J. M. Vaughn, whom he generously permitted me to include in this series. No persons with marked hyperplastic rhinitis or patients with definite seasonal hay fever were treated. However, patients with allergic nasal conditions which have not responded well to desensitization and other methods of control are being studied and will be reported on at a later date.

RESULTS

Nasal obstruction and associated symptoms were relieved completely in 66 per cent of the patients. The remaining 34 per cent were entirely or considerably relieved of nasal obstruction, and their postnasal drip and headaches were definitely diminished. The number of injections of the sodium morrhuate solution necessary to produce the desired effect

TABLE 1.—*Summary of the Number of Injections of Sodium Morrhuate Solution Into the Inferior Turbinates Required to Produce a Good Result*

Number of Injections Made Into the Inferior Turbinates	Percentage of Patients
1 injection into 1 turbinate *	13.8
2 injections into 1 turbinate **	5.2
3 injections into 1 turbinate	1.7
1 injection into each turbinate *	24.1
2 injections into each turbinate **	6.9
1 injection into 1 turbinate and 2 into other turbinate **	22.4
1 injection into 1 turbinate and 3 into other turbinate	8.6
2 injections into 1 turbinate and 3 into other turbinate	8.6
3 injections into each turbinate	3.4
3 injections into 1 turbinate and 4 into other turbinate	5.2
* 1 injection in 1 or both inferior turbinates	37.9
** 2 injections in 1 or both inferior turbinates	34.5
* and ** 1 or 2 injections in 1 or both inferior turbinates	72.4

TABLE 2.—*A study of the Individual Turbinates According to the Percentage of Turbinates Requiring One to Four Injections*

Name of Turbinate Injected	Percentage of Turbinates Requiring			
	1 Injection	2 Injections	3 Injections	4 Injections
Right middle turbinate	2.6	0.9	...	...
Left middle turbinate	3.5	2.6	0.9	...
Right inferior turbinate	27.8	9.5	6.0	...
Left inferior turbinate	17.4	18.2	7.8	2.6
Total turbinates treated	51.3	31.2	14.7	2.6

varied. One injection of 0.5 cc. frequently sufficed in one inferior turbinate, whereas two such injections might be required for the other inferior turbinate (table 1). One injection in one or both inferior turbinates was adequate for 37.9 per cent of those treated; 34.5 per cent required two injections in one or both inferior turbinates. Only 5.2 per cent required a fourth injection, and that in just one of the turbinates. As shown in table 1, 72.4 per cent of the patients required only one or two injections of the sodium morrhuate solution in one or both inferior turbinates. A study of both the middle and the inferior

turbinates injected revealed that 51.3 per cent had one injection and 31.2 per cent of the turbinates had two injections before the desired effect was produced (table 2).

PATHOLOGIC STUDIES

Biopsy specimens were taken from patients in whom both inferior turbinates appeared the same and shrank equally well. Tissue for microscopic comparison was removed from the middle third of one inferior turbinate before treatment and from the other inferior turbinate at varying intervals after completion of the treatment. Dr. G. H. Hauser, pathologist, cooperated in the histologic study.

The following characteristics were common to the tissues examined before injections of sodium morrhuate solution were made: (1) dilatation of the vascular sinuses, (2) hyperplasia of mucous glands, (3) chronic inflammation, as shown by small round cell infiltration, (4) many small blood spaces just beneath the epithelium in some specimens; polypoid tissue and cystic changes in mucous glands in others.

Biopsy specimens taken from one to five months after one or two injections had been made presented a microscopic picture of: (1) diffuse fibrosis obliterating many of the venous plexuses and a reduction in the size of the remaining vascular sinuses, (2) appearance of a "squeezing" effect on the glands by the surrounding fibrous tissue, (3) absence of cystic changes in the glands, (4) evidence of chronic inflammation at the end of five months, as indicated by some small lymphocyte infiltration, (5) absence of microscopic evidence of any deleterious effect on the epithelium of the mucous membrane.

The clinical benefit was brought about by the contracting effect of fibrosis, resulting in obliteration of the vascular spaces or marked reduction in their size. Since the postnasal drip ceased or decidedly decreased, the fibrosis must have reduced the activity of the mucous glands. The clinical benefit is directly proportional to the amount of fibrous tissue produced. However, each patient must be given individual treatment. One injection of sodium morrhuate solution in 1 person produces as much fibrosis as two or three injections in another. Therefore a physician should govern the number of 0.5 cc. injections at two week intervals according to the clinical results obtained. One must not overlook the possibility of overtreating patients in whom fibrous tissue is readily produced, although this never occurred in the series which I studied. If an overproduction of fibrous tissue occurred, such a hyperplastic condition would defeat the purpose of the therapy.

REPORT OF CASES

Protocols of some of the patients with interesting symptoms reveal the striking benefit received from treatment with sodium morrhuate. In order to save space and repetition, only brief mention will be made

of the anesthetization. In the cases herewith reported, the nose was shrunk from five to ten minutes after the injection of the solution was made, and the patient was advised to use nose drops of 1 per cent ephedrine in saline solution or a 0.5 to 1 per cent solution of neosynephrin every two to three hours for four days and then at longer intervals. Unless the physical examination and laboratory tests revealed something of importance, the results will be omitted from the case reports.

Case 1.—L. T., a Negress, aged 58, complained of nasal obstruction, cough and a postnasal drip, which she had noted for over fifteen years. She had been subject to frequent attacks of hoarseness and sore throat. A septum resection had been performed twelve years previously. The inferior turbinates were enlarged but shrank well with ephedrine. The mucous membrane was reddened and smooth. No pus was present in the meatuses. Chronic laryngitis and pharyngitis were present. On Oct. 5, 1939, tampons saturated with a 10 per cent solution of larocaine hydrochloride were applied around the left inferior turbinate for local anesthesia. An injection of 0.25 cc. of a 5 per cent solution of sodium morrhuate was made. The patient returned on October 18 and stated that she had been breathing much better through the left nostril for the past week. The right inferior turbinate was injected with 0.5 cc. of the solution. One week later 0.5 cc. of the solution was injected into the left inferior turbinate. The breathing had improved and the hoarseness and postnasal drip had diminished. A week later, November 2, 0.5 cc. was injected into the right inferior turbinate. When the patient returned two weeks later, there was no evidence of hoarseness, postnasal dripping or nasal obstruction. On Jan. 5, 1940, the nasal mucosa appeared normal, and there had been no recurrence of her previous symptoms. A biopsy specimen was taken from the right inferior turbinate. The patient was observed on April 4, five months after the last injection, and volunteered the information that she had felt better than for many years. The mucous membrane appeared clinically normal.

Case 2.—T. V., a Negress aged 24, complained of occipital, vertical and frontal headache, nasal obstruction and a postnasal drip, which had been rather constant for six months. She had been under treatment for over two months without obtaining relief. The history did not suggest allergy. The turbinates were reddened and swollen, but pus was not seen in the meatuses. Astringents reduced the size of the inferior turbinates. Marked opacity of the posterior ethmoid sinuses and a slight opacity of the left frontal sinus and the antrum were evident by roentgen examination. On Sept. 1, 1939 there was a mucopurulent discharge in the left middle meatus. The Proetz treatment was instituted and continued twice weekly for one month, with only slight improvement. On October 10, with a 2 per cent solution of butyn sulfate used as anesthetic, 0.33 cc. of a 5 per cent solution of sodium morrhuate was injected into the right inferior turbinate. When the patient returned one week later, there was less difficulty in breathing through the right nostril. There were less pain and headache. At this time, the left inferior turbinate was similarly treated. The headache, postnasal dripping and nasal obstruction were markedly diminished when the patient was observed the following week (October 24). The right inferior turbinate was still larger than it normally should be. A second injection of 0.5 cc. of the solution was made into it. The patient did not return for three months (Jan. 1, 1940), at which

time both inferior turbinates were somewhat turgescent. There was no complaint, but she stated that the right side of the nose occasionally was slightly stopped up. An injection of 0.5 cc. of sodium morrhuate solution was made into each inferior turbinate. An observation was made two weeks later. The turbinates were well shrunken, and the nasal mucosa appeared normal. The patient was seen on February 28 and on April 30. There had been no recurrence of symptoms. The patients own words were, "I'm feeling fine." The nasal mucosa appeared normal.

CASE 3.—S. M., a white woman aged 50, had had blockage of the nose for six years. Changes in temperature caused nasal obstruction and some sneezing. She also complained of frontal headaches and a feeling of congestion in the bridge of the nose. There had been anosmia for four months. She had been treated by shrinking methods for four years with little benefit, except for a few hours at a time. The turbinates were boggy and somewhat pale, and they shrank well with cocaine. Nasal smears did not show any increase in eosinophils. On Oct. 17, 1939 the left inferior turbinate was injected with 0.5 cc. of a 5 per cent solution of sodium morrhuate, after the application of a 2 per cent solution of butyn sulfate as an anesthetic. One week later there was less sneezing. The patient expelled much mucopurulent material from the left nostril for the first few days after the treatment. The left nostril did not become stopped up so much. The anterior portion of the right middle turbinate was injected with 0.13 cc. and the right inferior turbinate was injected with 0.33 cc. of the sodium morrhuate solution. The patient returned on October 30, feeling much better. The sense of smell had completely returned, and there was no nasal blockage or headache. This patient returned for observation about one month later, December 1, and stated that she was feeling fine. There was still a slight postnasal discharge. Five and one-half months after the last injection (April 8, 1940), there was no sneezing, headache or anosmia. She had been sleeping well and rarely had any nasal congestion. The mucosa appeared clinically normal.

CASE 4.—M. G., a Negress aged 33, complained of intermittent nasal obstruction, particularly at night, for eight years. She had been unable to sleep at night because of the difficulty in breathing. She had had frontal, temporal and occipital headache and pain in the postaural region and down the left side of the neck. These symptoms were more pronounced when the nose was stopped up. The pain and headache were relieved by cocainizing the sphenopalatine ganglion. A mucoid postnasal dripping was present. There was no history of allergy. Both inferior turbinates and the left middle turbinate appeared smooth, reddened and enlarged. They shrank well with cocaine. On Oct. 14, 1939, 0.5 cc. of a 5 per cent solution of sodium morrhuate was injected into the left inferior turbinate after tampons saturated with a 2 per cent solution of butyn sulfate had been used. One week later the headaches were less severe and less frequent. The right inferior turbinate was injected with 0.5 cc. of sodium morrhuate solution. When the patient returned a week later, November 29, the pain in the neck had disappeared, and she had experienced no more headaches. There was a marked reduction in the postnasal dripping. The following week, December 6, an injection of 0.5 cc. of the solution was made into the left middle turbinate. The right inferior turbinate was well contracted. Although the patient had definitely improved by the next week, December 13, 0.5 cc. of the solution was injected into the left inferior turbinate. All use of nose drops was discontinued the next week. The patient was observed about two and one-half months later, Feb. 29, 1940, at which time the mucous membrane appeared normal. The patient volunteered the statement, "I have had much treatment previous to this series of injections, with only

temporary benefit. I sleep well every night and feel better than I have for years." She was seen again about four months after the last injection, April 10, at which time the turbinates were of normal size and the mucous membrane appeared clinically normal. A specimen for biopsy was taken from the left inferior turbinate.

CASE 5.—L. P., a Negress aged 31, complained of nasal obstruction, which had been present for six months, marked postnasal dripping and recurrent attacks of sore throat. A tonsillectomy had been done in 1937. There was no allergic history. The left inferior turbinate was intumescent but shrank well with neosynephrin. Pus was not seen in the meatuses. An injection of 0.5 cc. of a 5 per cent solution of sodium morrhuate was made into the left inferior turbinate on Nov. 15, 1939, after the nasal tissues were anesthetized with tampons saturated with a 2 per cent solution of butyn sulfate. One week later the postnasal dripping was markedly reduced, and the patient was breathing better through the left nostril. When she returned the following week there was no postnasal dripping or nasal blockage. This patient was observed again about one month after the injection, at which time the pharyngitis and other previous symptoms had not recurred. When she was last seen, on Feb. 29, 1940, about two and one-half months after the injection, none of the symptoms had returned.

CASE 6.—J. A., a white woman aged 32, complained of frequent frontal headaches and postnasal dripping, which had been present for six years. The headaches and pains were worse in the left frontal and temporal regions and were more severe in the mornings. She also suffered from nasal blockage, which was usually intermittent but had been constant for the past three months. There was no history of allergy. Nasal smears showed many histiocytes and the presence of cell debris but no increase in the number of eosinophils. Roentgen examination revealed moderate thickening of the left posterior ethmoid sinuses and the left antrum. There was a small amount of mucus in the left middle meatus. The left inferior and middle turbinates were intumescent, reddened and smooth. They shrank well with ephedrine. On Nov. 8, 1939 the left inferior turbinate was injected with 0.5 cc. of a 5 per cent solution of sodium morrhuate after local anesthesia was obtained by the use of tampons saturated with a 2 per cent solution of butyn sulfate. A week later there were less headache and pain, and the patient was breathing better through the left nostril. At this time, 0.5 cc. of sodium morrhuate solution was inserted into the left middle turbinate. This patient returned about two and one-half months (Feb. 2, 1940) after the last injection. She had been entirely free from symptoms except for a slight pain in the left temporal region for the past week. After the nasal tissues were anesthetized with tampons saturated with a 10 per cent solution of larocaine hydrochloride, 0.25 cc. of sodium morrhuate solution was injected into the left middle turbinate. The next and last observation was made on March 30, approximately two months after the last injection and four and one-half months after the previous injection. There was no postnasal dripping and no difficulty in breathing through the left nostril. The patient had not used any nose drops for over four months, except for a few days after the last injection. She reiterated that before this treatment was instituted, severe headaches were present almost every morning. She seldom has a headache now, and when she does it is only slight and of short duration.

CASE 7.—E. J., a Negress aged 27, came to the clinic complaining of pain under the left eye, which had been present for two years, and an attack of hoarseness, which came on without any evidence of a "cold" and when there had not been excessive use of the voice. There was a history of recurrent attacks

of hoarseness. The patient had been having difficulty in breathing through the nose, particularly the left nostril, for two years. She complained of some dripping into the throat. There was no history or physical evidence of definite allergic manifestations. Examination revealed chronic laryngitis and postnasal dripping. Both inferior turbinates and the left middle turbinate were reddened, smooth and engorged. Pus was present in the left middle meatus. Roentgen examination of the sinuses showed marked opacity of the left antrum. A regimen of tincture of benzoin inhalations and voice rest and the use of nose drops were prescribed on Nov. 22, 1939. A specimen for biopsy was taken from the left inferior turbinate on December 1. The laryngitis was slightly improved. Four days later, 0.5 cc. of a 5 per cent solution of sodium morrhuate was injected into the right inferior turbinate, after the application of tampons saturated with a 2 per cent solution of butyn sulfate. On December 14, 0.5 cc. of sodium morrhuate solution was disseminated into the left inferior turbinate. There was no hoarseness. One week later there was little postnasal dripping. The pain under the eye had disappeared. After the application of tampons saturated with a 10 per cent solution of larocaine hydrochloride, 0.25 cc. of the sclerosing agent was injected into the left middle turbinate. When the patient returned for observation two weeks later, Jan. 4, 1940, there was no pus in the left middle meatus, no nasal obstruction, no postnasal drip or hoarseness and no pain in the infraorbital region. A biopsy specimen was removed from the right inferior turbinate. On May 8, more than five months after the last injection, the patient was still entirely free from her previous symptoms. The nasal mucosa appeared normal.

Case 8.—Z. R., a 56 year old white woman, complained of headache and of pain in the left frontal, temporal, occipital and postauricular regions, which extended down the neck. The duration of these complaints was twelve years. The pain and headache were relieved to some degree by cocainization of the sphenopalatine ganglion. These symptoms were aggravated when the nose was obstructed. Nasal congestion was worse in cool weather. There was some clear mucoid discharge from the nose. Ocular examination and correction of an error of refraction two months previously had not relieved the patient. The left middle turbinate was enlarged and pressing against the septum. The other turbinates were somewhat enlarged. They shrank well. On Dec. 7, 1939, the left middle turbinate was cocainized and 0.5 cc. of a 5 per cent solution of sodium morrhuate was injected into it at several places. One week later all pain and headache were gone. The patient had only one slight headache the succeeding week. One month after the injection, Jan. 6, 1940, the patient reported that she had experienced a slight occasional pain on the left side of the head during the preceding week. Tampons saturated with a solution of larocaine hydrochloride were used, and 0.25 cc. of sodium morrhuate solution was put into the left middle turbinate. Five days later, 0.25 cc. was injected into each middle turbinate, even though the patient was having no symptoms. Two months after the last injection, March, 5, the patient had not experienced a recurrence of the original symptoms. The middle turbinates were not enlarged. She returned on May 11, four months after the last injection. She had had an occasional headache, which was mild and lasted for only a short time. The other symptoms had not returned.

Case 9.—L. L., a 36 year old Negro, had had recurrent blockage of the nose for eighteen years, with alternate as well as bilateral obstruction at times. He had been having frontal headaches for many years. He also complained of a dripping into the throat. There was no history of sneezing or watery discharge

from the nose. A submucous resection had been done twelve years before. Perforation of the septum was present. The patient had been treated for these symptoms for several years with no permanent benefit. An ocular examination and refraction three months previously had failed to relieve him of his trouble. Examination revealed markedly engorged pale turbinates, which shrank well with cocaine. On Jan. 6, 1940, the left inferior turbinate was injected with 0.5 cc. of a 5 per cent solution of sodium morrhuate after tampons saturated with a solution of butyn sulfate had been applied. One week later the left inferior turbinate was smaller. Tampons saturated with a solution of larocaine hydrochloride were applied, and 0.5 cc. of sodium morrhuate solution was injected into the right inferior turbinate. The patient returned on February 3. Nasal congestion had been present for one week. Each inferior turbinate was injected with 0.33 cc. of the solution after the application of larocaine tampons. The patient experienced some dizziness for a few mniutes after the injection and perspired freely for five minutes. He did not complain of any discomfort in the head. The patient was observed three weeks later, February 24. Some postnasal dripping was the only remaining symptom. The turbinates were still somewhat turgescent. The right inferior turbinate was infiltrated with 0.5 cc. of sodium morrhuate solution. After three weeks, March 16, 0.5 cc. was injected into the right inferior turbinate, because the patient still had occasional blockage of the nose and slight postnasal dripping. A 2 per cent solution of butyn sulfate was used to induce anesthesia. He was observed two weeks later, March 30, and was breathing well through the nostrils. No more headaches had been experienced. The turbinates were not so pale and were slightly nodular. This patient was last seen seven weeks after the last injection, May 10, at which time there were no symptoms. The turbinates appeared more nearly normal than previously.

Case 10.—G. D., a 45 year old Negro, had had nasal congestion with headaches and nasal discharge for many years. A submucous resection had been done fifteen years before. A radical operation had been performed on the left antrum six years before. The administration of autogenous vaccine was started in January 1939 and was repeated every five days for five months, with no benefit. From July until November 1939, Dowling tampons and shrinkage methods were used with slight benefit. The symptoms were still present on Jan. 5, 1940. The turbinates were enlarged and rather pale. They shrank well with cocaine. Nasal smears did not show an increase in number of eosinophils but did show many plasma cells and evidence of chronic inflammation. A marked anterior and posterior nasal discharge was present. The roentgen examination revealed antral opacity and some involvement of the sphenoid sinus. The Wassermann and the Kahn test were negative. On January 8, after the application of cocaine-moistened tampons, the left inferior turbinate was injected with 0.5 cc. of a 5 per cent solution of sodium morrhuate. The right inferior turbinate was injected with the same amount of oil on the following week, after local anesthesia was obtained by the use of tampons saturated with a solution of larocaine hydrochloride. The patient returned three weeks later, February 8. Larocaine tampons were applied and 0.5 cc. of the oil was introduced into the left inferior turbinate. There was still some turgescence of the left inferior turbinate on February 20, at which time an injection of 0.5 cc. of the oil was made into it. When the patient returned one week later, the only remaining symptom was slight postnasal dripping. The patient was next seen on March 28, five weeks after the last injection. The postnasal dripping had ceased, and only one headache had occurred during this period. There was an occasional

slight blocking of the nose. Some turgescence of the inferior turbinates was still present. On April 13, tampons saturated with a solution of butyn sulfate were applied, and 0.5 cc. of sodium morrhuate solution was introduced into the right inferior turbinate. Two weeks later the left inferior turbinate was similarly treated. The patient was last observed on May 11 and was free of all symptoms. The mucous membrane appeared normal. He stated that he had been coming to the clinic for more than ten years and that this treatment had been more beneficial than any of the former procedures.

CASE 11.—C. H., a woman 52 years of age, complained of headache in the vertical and temporal regions, which was more severe at night. Frequently she experienced pain in the ears and tinnitis. Postnasal dripping, chronic sore throat and blockage of the nose were the other symptoms. The patient had suffered from these symptoms for nine years. There was no seasonal variation in their severity. Nasopharyngoscopic examination revealed some hypertrophy of the posterior portions of both inferior turbinates and edema around the orifices of the eustachian tubes; however, these turbinates shrank well with cocaine. Tampons saturated with a solution of butyn sulfate were used, and 0.5 cc. of a 5 per cent solution of sodium morrhuate was introduced into the left inferior turbinate on Jan. 8, 1940. Four days later the right inferior turbinate was similarly treated. On January 19 the nose was remaining open. The headaches were markedly improved, and the tinnitis, thumping and pain in the ears were gone. The left inferior turbinate was still somewhat turgescent, so 0.5 cc. of the oil was injected into it. The patient complained of some pain around the eye and the ethmoid regions after the injection. One week later the right inferior turbinate was treated with 0.5 cc. of the oil, and 0.25 cc. was introduced into the right middle turbinate. The patient was observed at intervals during the next three months. None of her former symptoms had returned. The patient was last seen on May 5. She said: "I have more pep and I feel better than I have for several years." The turbinates had returned to normal size.

The advantages of submucosal injections of sodium morrhuate solution over the other forms of treatment for chronic simple, hypertrophic and vasomotor rhinitis are:

1. No expensive equipment is necessary. One 5 cc. rubber-capped vial of the solution is more than sufficient for the entire treatment of a patient.
2. It is easily administered.
3. There are no ulcerations or adhesions.
4. The physiologic function of the nose is not disturbed except temporarily after the injection, as there is no distortion of the anatomic configuration which would interfere with the normal air currents. There is no deleterious effect or damage to the nasal mucous membrane.
5. There is comparatively little pain or discomfort after the injection.
6. If patients are properly chosen and the technic is followed as described herein, there should be no failures in relieving nasal obstruction.

SUMMARY AND CONCLUSIONS

The nasal obstruction in 58 patients with chronic simple, hypertrophic and vasomotor rhinitis was relieved by submucosal injections of a 5 per cent solution of sodium morrhuate. Associated symptoms, such as headaches, neuralgia of the sphenopalatine ganglion and postnasal dripping with chronic symptoms of the upper respiratory system, were markedly diminished or cured.

This form of therapy is a beneficial adjunctive in the treatment of long-standing refractory sinusitis because it reduces the chronically enlarged turbinates to normal size, thereby facilitating drainage and ventilation through the natural ostiums of the sinuses.

The procedures for proper choosing of patients and the technic of administration of the drug are described.

The length of time such treatment will relieve the symptoms has not been ascertained. Eight months have elapsed since the first patients of the series described in this paper received their last injection. So far, none of the patients has had a recurrence of nasal obstruction, and there is no clinical evidence of any deleterious effect of the drug on the nasal mucous membrane. This investigation is to be continued for another year. If at that time the patients remain well or improved, this form of treatment will have met the requirements of a desirable therapeutic agent in cases of chronic simple, vasomotor and hypertrophic rhinitis.

G. D. Searle & Co., Chicago, furnished the sodium morrhuate solution, in 5 cc. rubber-capped vials, for this investigation.

Abbott Laboratories, North Chicago, furnished the butyn sulfate for this study.

THE NASOPHARYNX

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In the office, the outpatient clinic and the hospital the lymphoid tissue in the pharynx, particularly that in the nasopharynx, plays a most important part in otolaryngology. Primary infections in this tissue extend to the sinuses, ears, larynx, bronchi and lungs. This is true particularly in children. Just where the primary focus for virus infections, such as the common cold, is I do not know, but it is certain that for pyogenic infections in the upper air passages the primary focus is lymphoid tissue. In the otolaryngologic outpatient departments of large hospitals there is an overwhelming demand for beds for patients whose tonsils and adenoids are to be removed, which cannot be met, especially the demand for free beds. Acute infections must be cared for in the hospitals, but it is the duty of otolaryngologists to foresee and treat conditions in their patients which they know by experience may lead to otitis and mastoiditis, deafness, sinusitis, asthmatic bronchitis and other systemic disturbances.

In treating ambulant patients, my associates and I, during the past twelve years, have used with great success a radon nasal applicator. The radon is of value in reducing the size of adenoids as an adjunct of the treatment of mouth breathing, recurrent nasopharyngitis, impairment of hearing due to tubotympanic catarrh, suppuration of the ears, infection of the accessory nasal sinuses and asthmatic bronchitis. The radon treatments do not supplant surgical operation, but are much more effective than sprays, nasal drops and local applications and in many cases have proved preferable to operation in tiding the patient over a difficult period. The radon applicator is particularly valuable in the treatment of young children, in reducing the size of lymphoid tissue in the nasopharynx and thus providing better breathing space and better drainage for the nasal cavities and eustachian tubes. Radium must not be used in the presence of acute nasopharyngitis. It has no inhibiting effect on the growth of bacteria.

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Infections of the upper respiratory tract, especially in children, result in hyperplasia and hypertrophy of lymphoid tissue. Even if the acute symptoms subside without systemic complications, the mechanical obstruction to breathing and to the eustachian tubes, the excessive mucous secretion and the lowered resistance of the patient prepare the ground for a recurrence of infection or a continuance of infection as a chronic condition. Even if all children who have such infections could be hospitalized and have their tonsils and adenoids removed, the end result would not always be satisfactory, since the abundance of lymphoid tissue in the oropharynx and nasopharynx makes impossible the removal of all of it at operation. In some children infections continue to recur after operation, and the posterior and lateral walls of the pharynx become studded with nodules of lymphoid tissue, a condition commonly described as "granular pharyngitis." Examination with a nasopharyngoscope shows the same condition more frequently and in a more advanced stage in the nasopharynx. The orifice of the eustachian tube is often completely or partially covered by this overgrowth of lymphoid tissue, which may also extend upward on the anterior surface of the sphenoid bone to the level of the middle turbinate.

An interesting fact about the nasopharynx was brought out by a study of 1,365 school children during the past year. All of the children were between the ages of 8 and 14 years. In 755 the tonsils and adenoids had been removed at various hospitals in Baltimore. As a rule, the tonsils had been cleanly removed, and in many the oropharynx looked normal, but more than 75 per cent had a marked recurrence of adenoid tissue in the nasopharynx. This was due not to incomplete operation but to the fact that in this area lymphoid tissue is an integral part of the mucous membrane. It cannot possibly be removed in its entirety unless the entire thickness of mucous membrane is taken out, a procedure which is manifestly impossible.

Therefore, in a large percentage of children whose tonsils and adenoids have been removed before the age of puberty, otolaryngologists must expect, look for and treat the recurrence of the tissue in the nasopharynx and pharynx if they are to obtain the best results. The proper treatment is irradiation, not a second operation. If the condition is overlooked or ignored, the ears are most likely to suffer. Acute attacks of otitis direct attention to the nasopharynx, but a chronic subacute tubotympanic catarrh may cause secondary changes in the middle ear that seriously and permanently impair the hearing. It is just as important for the otolaryngologist to examine the nasopharynx, the tympanic membranes and the hearing of children once or twice a year for the preservation of hearing as it is for the dentist to examine the teeth at intervals for their preservation. The first evidence of impaired hearing is a loss for high tones—those above the range of most of the

audiometers being used by otologists. The earlier the condition is recognized, the better are the chances of restoring the hearing and stopping the progress of the deafness. Progressive deafness usually begins with the high tones and advances by involving one tone after another toward the speech range (250 to 3500 double vibrations) and the low end of the scale.

When a child is found to have impaired hearing for high tones, hypertrophied adenoids that completely or partially cover the tubal orifices, recurring colds, repeated attacks of otitis media or a persistent discharge from the middle ear after paracentesis, the tonsils and adenoids may be removed—but with the full realization that the adenoids are likely to recur within a few weeks or months. The treatment for the recurrence is not a second operation, but irradiation. It is not the size but the location of lymphoid tissue that is important, and the location in and around the tubal orifices is such that the obstructing nodules of lymphoid tissue cannot be removed surgically without further damaging the tubes. Normal function of the eustachian tube is essential for good hearing. It is quite true that many children with enlarged adenoids or a recurrence after operation which obscures a view of the pharyngeal orifice of the tube have no impairment of hearing, but these children should be watched, and at the first sign of impaired hearing for high tones the nasopharynx should be irradiated. Repeated audiometric and nasopharyngeal examinations must be made and the radiation treatments repeated at intervals of a month until the orifices of the eustachian tubes are clearly seen. This is the simplest, safest and surest way to forestall the secondary changes in the middle ear that may cause the deafness to become permanent or progressive. Years of experience with this method of treatment have convinced us that if proper precautions are taken it is of great value and perfectly harmless.

Infected adenoids should always be removed surgically. The combination of operation and irradiation, or irradiation alone, prevents recurrences, or reduces the size and susceptibility to infection of the recurrent nodules of lymphoid tissue in the nasopharynx and pharynx. If radiation is employed, the otolaryngologist must realize that it does not destroy or permanently remove adenoid tissue. It merely slows up, or for a time stops, the reproduction of lymphocytes and in this way causes a gradual decrease in size. Usually two or three treatments are necessary to obtain the desired result, and they may have to be repeated once or twice each year until the child reaches the age of puberty. After this period there is a spontaneous regression of lymphoid tissue in the upper air passages in most children.

It is a comparatively simple matter to irradiate the nasopharynx with roentgen rays. The rays may be directed through the open mouth,

but this method of approach is difficult even in treating adults, because of the time required and the necessity for using a depressor to keep the tongue out of the field; it is even more difficult in the treatment of young children. If the rays are directed through the neck, the principle of cross firing must be employed. Since considerably more than half of the rays delivered at the surface of the skin are absorbed by the tissues of the neck before they reach the nasopharynx, it is evident that several portals must be used in order to prevent irritation to the skin. The fact that a large percentage is absorbed necessitates a dose on the surface of the skin much larger than that which is necessary to correct the lesion in the nasopharynx, for which the treatment is given. This excessive dose subjects the centers of ossification for the growing bones of the face and skull to a large amount of radiation, which may do harm in a child and is of no value in the treatment of the primary condition in the nasopharynx. Furthermore, it is difficult to maintain young children in a position for accurate cross firing for the duration of the treatment. Successful results in shrinking lymphoid tissue in and around the orifice of the eustachian tube and restoring the hearing cannot be expected unless the central beam in cross firing actually passes through the area where treatment is most needed. Finally, roentgen treatments should not be carried out by any one except an experienced technician or radiologist.

In contrast, the radon nasal applicator can be used by the otolaryngologist, who tests the hearing, examines the nasopharynx and places the applicator on the exact spot where the treatment is most needed. The applicator is small enough to pass along the floor of the nose of an infant. No anesthesia is necessary. Any amount of radon up to 1,200 millicuries can be used in this small applicator, but on account of the bulk, not more than 50 mg. of pure radium can be put in a tube of this size (2 cm. in length and 1.5 mm. in diameter). The number of minutes required for an adequate treatment depends on the number of millicuries or milligrams of active material in the applicator. With 1,000 millicuries of radon the time would be only two minutes on each side; with 100 millicuries, twenty minutes, and with 50 mg. or millicuries of radium salts, forty minutes. With a strong applicator, one two minute treatment is equivalent to the fractional doses of roentgen, which must be extended over a period of two or three weeks.

In conclusion I wish to stress four points:

1. The recurrence of adenoids after operation on young children is so common that it must be regarded as normal.

2. This recurrent lymphoid tissue may impair the function of the eustachian tubes and cause a low grade tubotympanic catarrh, which may lead to chronic progressive deafness.

3. Recurrent or hyperplastic nodules of adenoid tissue in and around the pharyngeal orifice of the eustachian tube are so located that they cannot be removed surgically.

4. Lymphoid tissue is so sensitive to radiation that the dose employed in the treatment of the nasopharynx is far below the amount that could cause any irritation or injury to the mucous membrane or surrounding structures. Scar tissue, dryness of the nose or throat and atrophic changes in the nasal mucosa do not follow treatment of the nasopharynx with radon, provided the proper dose is given at the proper intervals. This statement is based on my observation of hundreds of children. Many of them have been followed for ten years or more. If used carelessly or if entrusted to a technician, radium therapy may be extremely dangerous. It should never be given in the presence of acute nasopharyngitis. The object of the treatment is to reduce the size of lymphoid tissue. It has little or no direct action on bacteria. Radiation is of no value in the treatment of chronic suppuration of the middle ear, otosclerosis or any form of inner ear or nerve deafness. It is used for the sole purpose of reducing obstructing nodules of lymphoid tissue, decreasing the secretion of mucus and restoring the normal ventilating function of the eustachian tubes. For this reason radiation is more often beneficial for children than for adults. To obtain the best results it must be used before changes in the middle and inner ear, secondary to partial tubal obstruction, have irreparably damaged the structures of the middle and inner ear.

SPREADING OF VACCINE IN NASAL VACCINATION

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The upper respiratory tract presents an ideal absorptive surface for the application of antigenic material. Krishnamurthy [1] found that the absorption of bacteria in the tracheal mucosa of immunized animals takes place at a slower rate than in normal animals. Maugeri [2] studied the penetration of the tracheal mucosa by anthrax bacilli, Bacillus subtilis, sarcinas and staphylococci. The organisms were found in the epithelium within fifteen minutes, and after six hours they had disappeared entirely. Nasal immunization has the advantage over the ordinary methods of immunization in that a lesion is not produced by the inoculation. Although nasal immunization has been tried over a period of years (Bailey,[3] Bull and McKee,[4] Sabin,[5] Sewall,[6] Walsh and Cannon [7] and others), it has had only a limited practical application. The main reason for the limited use of this procedure has been the possible danger of causing pneumonia. In fact, in a number of cases observed, pneumonia developed after nasal injection (Bull and Bailey [8]). On the other hand, to date no definite information exists as to the extent to which a fluid spreads when applied nasally. Mullin and Ryder [9] instilled india ink into the nostrils of rabbits maintained in a recumbent position and found that it readily spread to the terminal bronchi and alveoli. Corper and Robin [10] observed that india ink instilled intranasally in anesthetized rabbits and dogs which were kept in a horizontal position reached the upper lobes of the right or left lung, depending on the side on which the animal was lying. If the animals were in a vertical position when the dye was instilled, it spread to the

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lower lobes. Corper and Robin [10] were of the opinion that the localization of aspirated fluids in the lungs depended mainly on the gravity and inspiratory suction. This viewpoint was shared by Cannon and Walsh,[11] who instilled india ink or trypan blue into the nostrils of 28 rabbits and 3 guinea pigs. In 17 of 20 rabbits kept with the head slightly raised, the dye reached the upper lobes of both lungs. In 5 animals kept on the side in horizontal position, the ink reached the upper lobe of the lung on the dependent side. In 6 animals which were kept in vertical position, the ink spread to the lower lobes. Even in unanesthetized animals kept in horizontal position the ink usually entered the lungs. These interesting observations give rise to some more important considerations, namely, (1) whether nasal instillation can be carried out without the fluid reaching the bronchi and (2) to what extent the results obtained on animals can be applied to man. In order to determine how to prevent the development of pneumonia after nasal inoculation and to ascertain the surface area over which the inoculated material may spread, the studies to be reported here have been undertaken. The technic used and the observations made are given.

ORIGIN OF STUDIES

A large number of rabbits have been inoculated orally with various antigenic materials. The animals resisted the intrusion of the droppers in the mouth by biting off the ends of them and thus injured the tongue and other soft parts of the mouth. The bleeding which resulted induced the animals to spit out the blood and, with it, more or less of the antigenic material. Even without bleeding, most of the animals tried to expel the antigen. These circumstances made it difficult to determine the amount of antigenic material which actually entered the esophagus. Therefore an attempt has been made to use the droppers nasally instead of orally. It was observed that some rabbits tried to blow the material back, especially when a pipet was used. The back blowing was prevented by keeping the rabbit's mouth open during inoculation. The nasal inoculation permitted an exact measurement of the injected material.

TECHNIC EMPLOYED IN EXPERIMENTATION ON RABBITS

Rabbits weighing 5 to 6 pounds (around 2.5 kg.) were used principally in the animal experiments described here; as droppers, 1 cc. pipets with perfectly smooth ends were employed. They were inserted carefully by pushing them along the septum after previously widening the external nares. Under no circumstances were they pushed deeper than 1.5 cm. These precautions were found essential in order to avoid injuring the nasal mucosa. The trauma was frequently followed by suppuration, which rendered further inoculation difficult. Among the

11. Cannon, P. R., and Walsh, T. E.: J. Immunol. **32**:49, 1937.

sprays manufactured by the DeVilbiss Company, those of type 25 were found to be most satisfactory, in shape as well as in the facility with which they can be cleaned. The spraying end was filed down so as to resemble the end of the dropper. In order to assure the instillation of approximately equal amounts, the fluid was sprayed once before and once after the inoculation. One additional requirement was that the rabbit be kept in a fixed position, in order to avoid unnecessary injuries. This was done by means of various types of rabbit boards or by holding the hindlegs and the head firmly.

EXPERIMENTS

In tracing the spread of antigenic fluid, the following aniline dyes were experimented with: gentian violet, dahlia violet, methyl violet, crystal violet, and methylene blue. One per cent gentian violet in a 10 per cent alcoholic solution was predominantly used. Brilliant green had to be abandoned on account of its toxicity for the animals. Half an hour after the application of the dye solution, the animals were killed, dissected and examined for the spread of the dye. In doubtful cases frozen sections were prepared and the respective dyes traced in the unstained tissues.

REPORT OF EXPERIMENTS ON RABBITS

EXPERIMENT 1.—A rabbit in standing position with hindlegs tied and head held firmly received from a 1 cc. pipet 1 drop of gentian violet. It was allowed to rest for one-half hour and was then killed and examined. The dye stained both nasal cavities, the pharynx, the mouth, including the palate and tongue, and the entire respiratory tract, down to the smallest bronchi and alveoli. Portions of the lungs were grossly stained, as ascertained by examination of the surfaces and cross sections. The entire esophagus, large portions of the stomach and, in some instances, of the duodenum were also stained. The use of several drops of gentian violet did not essentially alter the result, except that the excess of the dye ran from the nostrils and mouth and that the staining effect on the described tissues was intensified, especially in the stomach and duodenum.

The findings in the respiratory tract show plainly why a vaccine applied orally by the drop method could easily produce considerable irritation in the smaller bronchi and alveoli and could engender a type of pneumonia commonly known as aspiration pneumonia. This experiment further shows how a small amount of fluid can be spread over relatively large surfaces sufficiently to cause pneumonia. These findings confirm previous reports of the development of pneumonia after nasal instillation, which theoretically should occur in every case of nasal inoculation by the drop method. The fact that the disease does not always occur is a positive proof that other factors besides the presence of an irritant fluid in the bronchi are necessary to produce the disease.

EXPERIMENT 2.—This experiment was carried on in the same way as the preceding one except that the mouth of the rabbit was kept open during inoculation. The observations at autopsy were essentially the same as those following the

experiments in which the mouth of the rabbit was closed; the results were not changed if several drops were instilled instead of 1.

These observations indicate that the opening of the mouth during inoculation by the drop method has no effect on the spread of the dye.

EXPERIMENT 3.—The rabbit was kept in the same position as that in experiment 1. A DeVilbiss spray was used instead of a dropper, and only one inoculation was applied. The conditions seen at autopsy were the same as in experiment 1 except that the staining of the upper respiratory tract extended definitely only as far as the vocal cords and only faintly 1 to 2 cm. below the vocal cords. This observation was further substantiated by a subsequent histologic examination of the frozen sectioned tissues. These findings were constant in all 18 rabbits treated in this manner.

The results of this experiment indicate that vaccine can be safely applied nasally by the use of the spray without any danger of producing aspiration pneumonia. These results are considerably changed if several sprays are applied in rapid succession. The excess fluid, which is not absorbed, runs downward in the same way as if the drop method had been employed, staining the smallest bronchi and alveoli. This would indicate that large amounts of a vaccine can be satisfactorily applied nasally by means of a spray if the vaccine is used in a more concentrated form and if a single application is limited to 1 spray. It is obvious that if immunization by means of a nasal spray gives the same, or nearly the same, results as that by the subcutaneous method, the nasal spray procedure is preferable.

EXPERIMENT 4.—The rabbit was treated in the same way as the one in experiment 3 except that the mouth of the animal was kept open during the spraying. The observations made at autopsy differed only slightly from those in the preceding experiment.

The outcome of the staining shows that only slight differences occur when the mouth of the experimental animal is kept open during spraying.

EXPERIMENT 5.—The rabbit was held in the same manner and in the same position as the one in experiment 2. One drop of gentian violet was applied by means of a dropper, orally instead of nasally. The staining results were the same as those in experiments 1 and 2 except that only the posterior portions of the nasal cavities were stained slightly. Several drops produced the same effect except that the staining of tissues was more intensified.

These findings further indicate that oral immunization by means of a dropper presages the same dangers as those observed with the nasal drop method.

EXPERIMENT 6.—The rabbit was held in the same position as in the preceding experiment. The stain was instilled in the mouth by means of a spray. Only 1 spray was applied. The obtained results resembled closely those described in experiment 4 except that the nasal cavities remained unstained. Several sprays in rapid succession produced conditions identical with those described in experiment 5.

These observations further indicate that the oral and nasal spray methods offer the same beneficial results if the inoculation is limited to a

single spray at each inoculation. The nasal spray method would be preferable to the oral spray procedure when applied to human beings.

EXPERIMENT 7.—The rabbit was held in the same position as in experiment 3 and a freshly prepared 1 per cent solution of brilliant green instead of gentian violet was used as a dye. The autopsy revealed, contrary to expectation, the same conditions as those seen in experiment 1.

Subsequently it was found that other dyes with a toxic effect on animals produce similar results. Bull and Bailey [8] observed the development of lobar pneumonia, fibropurulent pleurisy and pericarditis after the nasal instillation of brilliant green in rabbits.

EXPERIMENT 8.—In order to determine what influence the position of the rabbit exerts on the experimental results, experiments 1 to 6 were repeated with the animals lying on their backs. It was found that the change in the position did not affect in any way the previously recorded results except for the involvement of the lungs.

The results of this experiment indicate that the position of the rabbit at the time of inoculation had no important influence on the results.

EXPERIMENT 9.—This experiment was conducted with 4 dying rabbits. Rabbit 1 received 1 drop of gentian violet nasally, rabbit 2 received 1 spray nasally, rabbit 3 received 1 drop orally and rabbit 4 received 1 spray orally. Rabbits 1 and 2 showed the same results as the rabbit in experiment 1. Examination of rabbit 3 and 4 revealed staining identical with that seen in the rabbit in experiment 5.

The results point definitely to the influence of the epiglottis in these experiments.

EXPERIMENT 10.—This experiment was conducted with 4 dead rabbits. Rabbit 1 received 1 drop of gentian violet nasally, rabbit 2 received 1 spray nasally, rabbit 3 received 1 drop orally and rabbit 4 received 1 spray orally. The results differed considerably from those described in the preceding experiment. In all four rabbits the collapsed esophagus remained unstained, while the open epiglottis allowed the fluid to pass through the open vocal cords, reaching the smaller bronchi and alveoli.

EXPERIMENT 11.—One rabbit received a nasal spray of 10 per cent procaine hydrochloride and then, fifteen minutes later, 1 nasal spray of gentian violet. Half an hour after the last spray it was killed and dissected. Another rabbit received an oral spray with procaine hydrochloride and, after fifteen minutes, an oral spray with gentian violet. The first rabbit showed staining resembling that in experiment 1. The second rabbit showed results identical with those produced on the rabbit described in experiment 6.

Both observations indicate plainly that with the nonfunctioning of the epiglottis the spray and drop methods (nasal as well as oral) have the same effects.

REPORT OF EXPERIMENTS ON HUMAN BEINGS

EXPERIMENT 1.—The initial experiments showed that the results obtained in rabbits did not correspond to those obtained in human beings. When methylene blue was used, the stain of the palate, the pharynx and the posterior dorsum

of the tongue faded rapidly. This applied likewise to the external surface of the epiglottis. Methylene blue was subsequently discarded as unsatisfactory.

EXPERIMENT 2.—One spray with 1 per cent gentian violet was administered to a human subject. Subsequently inspection revealed that the respective nasal cavity was well stained and that little of the dye passed the posterior nares, staining portions of the pharynx only slightly. The same results were obtained when 1 drop of the dye was instilled by means of a dropper.

These results plainly demonstrate that 1 drop or 1 spray suffices to stain a nasal cavity thoroughly. It has been shown already that a vaccine can be easily concentrated in such a manner that 1 drop or 1 spray may contain the desired amount of killed bacteria. One drop or 1 spray of a vaccine represents a safe amount, which can be applied without any danger of its passing to the larynx and causing respiratory disturbances of any kind.

EXPERIMENT 3.—Six to 8 sprays of dye were instilled in a nostril of a subject, and after a few minutes the staining effects were examined. It was found that the nostril was intensely stained, and, to a lesser degree, the pharynx, most of the palate, the posterior part of the dorsum of the tongue, the external surface of the epiglottis and the rim on the inside. The vocal cords remained completely unstained. This experiment was repeated on several other persons with the same results.

Here a staining area entirely different from that in experiment 2 was observed, with the stain extending in a slight degree to the inside of the epiglottis. Although the dye did in no instance reach the vocal cords, the margin of safety appeared considerably decreased as compared with the results obtained by the use of 1 spray or 1 drop.

EXPERIMENT 4.—Six to 8 drops of 1 per cent gentian violet was administered nasally to a number of persons, and after several minutes the staining effects were recorded. The results obtained by the use of several drops corresponded in all essentials to those observed after the use of several successive sprays. They included also the same dangers that were observed when several sprays were used.

EXPERIMENT 5.—Several subjects received 1 spray of 1 per cent gentian violet orally and were subsequently examined for the staining effects. The results were identical with those described in experiment 1 except that the mucosa of the nasal cavities remained unstained and the external surface of the epiglottis showed a slight degree of staining.

EXPERIMENT 6.—Several persons received 1 drop of 1 per cent gentian violet orally. After ten minutes they were examined, and the stained area corresponded essentially to that in the preceding experiment.

EXPERIMENT 7.—Several sprays of gentian violet were administered orally, and the staining effects after the expiration of several minutes were traced and recorded. It was found that the pharynx, the palate, the tongue and the external surface of the epiglottis were intensely stained and that the vocal cords remained unstained. The same results were obtained when several drops of the dye were instilled orally.

These results indicate that the application of several sprays or drops nasally or orally always implies the danger that a small fraction of the fluid may pass the vocal cords and cause foreign body irritation in the bronchi.

SUMMARY

It was always the objective of any kind of immunization to attain the maximum effect under the least disadvantageous circumstances. In intravenous immunization the response to the introduction of antigenic material has been usually most rapid and effective. At the same time this response has been preceded or accompanied by general symptoms of varying degrees, but always more severe than those observed in connection with other forms of immunization. The subcutaneous method has been preferred because the reaction is less rapid and the general symptoms milder and because larger amounts of the antigen can be injected with much less risk. Nevertheless, the subcutaneous immunization is always followed by some general symptoms. In addition, it represents a surgical step in which the rules of asepsis must be observed almost as rigidly as in any other operation. Those who had the opportunity to perform wholesale immunizations by the subcutaneous method during the World War will recollect the complications they encountered when working under unfavorable conditions. Even in the vaccination of a civil population on a large scale, it is not always possible to work at all times under strictly aseptic conditions. This may be especially true in rural communities in times of epidemic. Therefore it is conceivable that any new method should be preferred which is capable of producing as efficient results as the subcutaneous procedure and at the same time is not hampered by its disadvantages.

Although nasal inoculation has been tried over a period of years, it has not attained practical importance to date. One of the discouraging factors has been that occasionally pneumonia has been observed in connection with nasal inoculations.

It has been the objective of the studies reported here to determine the mode by which a nasally injected fluid spreads and to ascertain whether possibilities exist to avoid development of pneumonia in connection with this method of immunization. It has been established, as will be shown in subsequent papers, that the nasal mucosa presents an excellent absorptive surface for antigens and antibodies. The inoculation is easily performed without injury. The favorable response to the formation of antibodies will be shown subsequently. The absence of general symptoms after inoculation is another advantage. The risk of the development of pneumonia after nasal inoculation is negligible if the amount of antigenic material used in instillation is limited to 1 spray or 1 drop. This safety applies in cases of human beings equally whether the spray method or the drop method is employed. The results obtained

in the inoculation of rabbits were entirely different from those in the inoculation of human beings under the same experimental conditions. Several factors account for this:

1. The nasal mucosa of a rabbit presents a much smaller surface area than that of a human being.

2. The human epiglottis appears to be a more perfect mechanism, which, under normal conditions, easily prevents any fluid from entering the larynx, regardless of the amount injected nasally.

3. In the inoculation of rabbits it is only under the most favorable conditions that the fluids from a spray do not spread farther downward into the larynx than the vocal cords or the region immediately below. In all other circumstances it spreads, even when limited to 1 drop, to the smallest ramifications of the bronchi and alveoli. This has been confirmed by gross and histologic examinations.

The rabbits did not appear to be suitable experimental animals from which to draw conclusions as to the possible effects of the nasal application of antigenic materials on human beings. In human subjects most of the fluid remained in the nasal cavity, and it was plainly demonstrated that the nasal cavity was deeply stained, while other tissues were only slightly stained. In rabbits there was hardly any difference in intensity of staining between nasal mucosa and other involved tissues. These results plainly indicate that 1 drop or 1 spray represents a safe amount of antigenic material for use on human beings but not for use on rabbits.

CONCLUSIONS

1. The human nasal mucosa presents a large and easily accessible absorptive surface for antigenic material.

2. If the administered material is limited to 1 drop or 1 spray, it can be safely applied without any danger of causing aspiration pneumonia.

3. Nasal inoculation has the advantage over other methods of immunization that it does not cause any injury.

4. The recommended amount of 1 drop or 1 spray of the antigenic material suffices to spread thoroughly and chiefly over the entire nasal mucosa, according to the studies made with dyes.

5. One per cent gentian violet is satisfactory for use in the study of the spread of antigenic material by the nasal route.

6. Rabbits are not suitable for the study of nasal immunization as applied to human beings.

7. The human epiglottis represents a highly perfected mechanism, which normally prevents any material from entering the larynx. The epiglottis of rabbits offers much less protection.

The human subjects used in this experimental work were patients of Dr. T. S. Kammerling, of the Chicago Eye, Ear, Nose and Throat Hospital.

REACTIONS OF LARYNGEAL TISSUES FOLLOWING EXTENDED FRACTIONAL ROENTGEN IRRADIATION

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INTRODUCTION

During the past decade Coutard's [1] method of protracted fractional irradiation has made it possible to treat neoplasms of the larynx, without serious or fatal consequences, with doses many times those given in earlier years. However, because Coutard's method is a principle rather than a well defined technic and is undergoing, as experience dictates, many adaptations, the question of surgical procedure versus irradiation in the treatment of laryngeal neoplasms remains at present sub judice. The number of patients treated by radiation and followed for five to ten years is small, and opinion on the effect of roentgen treatment is therefore based on evidence insufficient in many respects.

Despite the fact that it is now possible to give sufficient roentgen radiation, following the principle of Coutard, to cause disappearance and clinical cure of carcinoma of the larynx in many cases, few studies of radiation effects on laryngeal tissues appear in the literature. Aside from the publication of Kahlstorf and Zuppinger,[2] a few short scattered remarks and the report of Windholz,[3] based on examination of 10

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1. Coutard, H.: (*a*) Zusammenfassung der Grundlagen der röntgentherapeutischen Technik der tiefgelegenen Krebse, Strahlentherapie **37**:550, 1930; (*b*) Roentgentherapy of Epitheliomas of the Tonsillar Region, Hypopharynx and Larynx from 1920 to 1926, Am. J. Roentgenol. **28**:313, 1932; (*c*) Principles of X-Ray Therapy of Malignant Diseases, Lancet **2**:1, 1934; (*d*) Ueber Roentgentherapie der Pharynxcarcinome, Strahlentherapie **52**:1, 1935; (*e*) Cancer of the Larynx: Results of Roentgentherapy After Five and Ten Years Control, Am. J. Roentgenol. **40**:509, 1938.

2. Kahlstorf, A., and Zuppinger, A.: Unsere Erfahrungen mit protrahiert fraktionierter Roentgenbestrahlung nach Coutard, Strahlentherapie **38**:199, 1930.

3. Windholz, F.: Zur Strahlentherapie der Kehlkopfcarcinome. Histologische und experimentelle Untersuchungen zur Röntgenbestrahlung von Kehlkopfcarcinomen nach Coutard, Monatschr. f. Ohrenh. **68**:1250, 1934.

larynxes, the majority of which were given overdoses, there are no studies of the effects of radiation on neoplasms and normal tissue of the larynx. While the cases mentioned are of some interest, they do not answer the question of what occurs when the larynx has been irradiated according to the present view of correct dosage.

A consideration of the histologic changes caused by the present method is important, for such information may help to confirm or refute opinion concerning the effects of radiation on the larynx and may, in addition, indicate the direction in which further improvement of therapeutic methods may be expected. It was with the purpose of determining these histologic effects of radiation that our study was made.

METHOD

Representative sections from various portions of the larynx of 16 patients were examined, some being taken from the pathologic anatomic collection of the otolaryngologic department of the Presbyterian Hospital–Columbia Medical Center in New York (cases 1, 3, 8, 9, 12, 13 and 14) and the rest from the otolaryngologic department of the University of Vienna Medical School (cases 2, 4, 5, 6, 7, 10, 11, 15 and 16).

The larynxes represented were divided roughly into two groups: (1) those examined within from four to fifty-one days after the termination of radiation treatment and considered, in this study, to show the early reaction of the mucosa and epithelium, and (2) those examined from three and one-half months to two and one-half years after the termination of radiation treatment and considered to show the late reaction of the mucosa and epithelium. Because it is more difficult to divide the effects on tumor tissue in this manner, no such arbitrary classification was used when these effects were considered.

A summary of the clinical records of the patients from whom the first group of larynxes were taken (cases 1 to 8) is found in table 1, and a summary of the radiation treatment received by each patient, in table 2.

HISTOLOGIC OBSERVATIONS ON LARYNXES EXAMINED BETWEEN FOUR AND FIFTY-ONE DAYS AFTER THE TERMINATION OF RADIATION TREATMENT

The first 8 larynxes showed the stages of early reaction of the tissues to irradiation. A summary of the epithelial changes may be found in table 3. One, considered to be in the earliest stage of such changes, showed unaltered epithelium, dilatation of the superficial vessels of the subepithelial tissues, exudation of serum into the tissues and beginning emigration of leukocytes. In 2 (cases 2 and 3) there were mild changes in the stratified squamous epithelium in the form of decrease in the number of layers without increase in superficial desquamation of the stratified squamous epithelium.

The ciliated epithelium showed progressive circumscribed or diffuse transition to stratified squamous epithelium, which was most marked in

TABLE 1.—*Clinical Data on First Eight Cases (Sections from Larynx Examined Within Four to Fifty-One Days After End of Irradiation)*

Case	Sex	Age, Years	Clinical Course	Observations at Autopsy
1	M	52	Hoarseness 2 mo.; radiation 5/22/33 to 5/27/33; laryngectomy 5/31/33	No autopsy (operative specimen showed involvement as noted in table 5)
2	M	65	Pain on swallowing 3 mo.; 3/12/37 removal of cervical glands; 3/25/37 to 4/17/37, radiation; 5/2/37 death from septicemia	Larynx similar in localization to that in table 5; metastasis to left anterior cervical glands; purulent thrombophlebitis, left saphenous vein
3	M	62	(*a*) Hoarseness 2 mo.; 10/13/31 to 11/16/31, radiation and gradual subsidence of tumor; membrane at base of epiglottis in 22 days, spreading in 7 days around periphery of larynx and into interior; gradual disappearance after 1 mo.; edema for 2 mo. (*b*) 6/14/34, pain and expectoration of blood; ulceration in right vallecula; 6/21/34 to 7/12/34, radiation; membrane formation of fauces, epipharynx and uvula in 2 wk.; disappearance of this and ulcer in 1 mo. (*c*) 10/1/34, discomfort and new ulceration in vallecula; 7/18/35 to 7/25/35, radiation; gastrostomy, 7/29/35; death 8/7/35	Lobular pneumonia, both lower lobes; ulceration of vallecula and infiltration of base of tongue; laryngeal mucosa bluish gray; vocal cords edematous
4	M	68	Hoarseness 5 yr.; 7/6/34 to 8/1/34, radiation; regression of tumor in 4 weeks; membrane formation beginning 14 days after start of radiation treatment involved piriform sinus and right ventricular fold; death 8/31/34	Emphysema of both lungs; hypertrophy of right ventricle; brown atrophy of myocardium; ventricular bands and vocal cords thickened; ulceration of left vocal cord
5	M	50	Hoarseness 4 yr.; history of syphilis; 8/4/36 to 9/8/36, radiation; rapid regression of tumor; membrane on ventricular fold in 10 days, followed by membrane on posterior laryngeal wall; death 9/12/36	Atherosclerosis of coronary vessels; marked atherosclerosis and mesaortic changes of ascending aorta and arch; larynx: edema of right aryepiglottic fold; thickening of vocal cords and ventricular bands; perichondritis; abscess cavity in right thyroid cartilage region
6	M	75	Hoarseness 1 yr.; 12/14/36 to 1/18/37, radiation; rapid regression of tumor; membrane formation on tumor surface, ventricular folds and vocal cords, later in interior of larynx, aryepiglottic folds and piriform sinuses; death 2/2/37	Lobular pneumonia, both lower lobes; mucous membrane of epiglottis, aryepiglottic folds, vocal cords and ventricular bands edematous and gray-white
7	M	74	Hoarseness 6 weeks; 5/14/37 to 6/16/37, radiation; regression of tumor in 3 weeks; membrane formation starting 19 days after beginning of radiation treatment involving aryepiglottic folds, piriform sinuses, vallecula and posterior laryngeal wall; 3 weeks later, gradual disappearance; death 7/5/37	Embolism, main branch of right pulmonary artery; empyema of right pleural cavity; pneumonic foci right lower lobe; mucous membrane of larynx slightly edematous
8	M	62	Hoarseness 14 yr.; 11/5/36 to 12/2/36, radiation; decrease in tumor size with surface membrane reaction; bronchopneumonia and then death 3/14/37	Bilateral bronchopneumonia and bilateral organized fibrinous pleurisy; laryngeal mass involving ventricular band and adjacent ventricle on right, extending up to base of epiglottis

TABLE 2.—*Data on Roentgen Therapy in Sixteen Cases*

Case	Date	Skin Dose (r)	Fields	Size of Field	Dose (r) per Field	Dose (r) per Minute	Dose (r) per Sitting	Data
1	5/22/33 5/27/33	2,400	2 lateral cervical	8 x 10 cm.	1,200	10.0	400	kv. 190; a. s. d. 50 cm.; ma. 8; filter 1.86 mm. Cu. and 1 mm. Al.
2	3/25/37 4/17/37	5,400	Right lateral cervical	140 cm.²	5,400	6-7.5	150	A. s. d. 40 cm.; H. V. L. 1.2
3	10/13/31 11/16/31	9,000	2 lateral cervical	6 x 8 cm.	4,500	12	300	kv. 200; a. s. d. 50 cm.; ma. 8; filter 1.86 mm. Cu. and 1 mm. Al.
	6/21/34 7/12/34	5,400	2 lateral cervical and posterior	8 x 10 cm. 6 x 8 cm.	1,800	12	300	kv. 190; a. s. d. 50 cm.; ma. 8; filter 1.86 mm. Cu. and 1 mm. Al.
	7/18/35 7/25/35	1,750	Right lateral and posterior	8 x 10 cm. 6 x 8 cm.	1,000 750	15	250	kv. 200; a. s. d. 80 cm.; ma. 25; filter 1 mm. Cu. and 1 mm. Al.
4	7/16/34 8/ 1/34	6,700	2 lateral cervical	85 cm.²	3,350	7-8	240- 390	A. s. d. 40 cm.; H. V. L. 1.0
5	8/ 4/36 9/ 8/36	8,050	2 lateral cervical	100 cm.²	4,025	6-8	150-	A. s. d. 40 mm.; H. V. L. 1.2
6	12/14/36 1/18/37	7,800	2 lateral cervical	50 cm.²	3,900	5-8	300 400	A. s. d. 40 cm.; H. V. L. 1.2
7	5/14/37 6/16/37	7,650	2 lateral cervical	80- 110 cm.²	4,200 r. 4,350 l.	6-7	200- 400	A. s. d. 40 cm.; H. V. L. 1.2
8	11/ 5/36 12/ 2/36	4,800	2 lateral cervical	8 x 10 to 10 x 10 cm.	2,400	21	150- 300	kv. 200; a. s. d. 50 cm.; ma. 25; filter 2 mm. Cu. and 1.25 mm. Al.
9	6/27/29 7/27/29 and 9/16/29 10/19/29	4,800	2 lateral cervical	10 x 10 cm.	2,400	..	180	kv. 158, later 180; a. s. d. 50 cm.; ma. 4; filter 0.53 mm. Cu. and oil
10	3/20/35 4/17/35	7,700	2 lateral cervical	180 cm.²	3,600	5-8	250- 100	A. s. d. 40 cm.; H. W. S. 1.2
	4/24/35 4/26/35	500	Submental	180 cm.²				
11	9/ 3/37 10/ 9/37	8,050	2 lateral cervical	50 to 120 cm.²	6,350 r. 1,700 l.	5-7	150- 200	A. s. d. 40; H. V. L. 1.2
12	1/27/36 5/21/36	4,400	2 lateral cervical	6 x 8 cm. 8 x 10 cm.	2,400 r. 2,000 l.	20.6	200- 250	kv. 200; a. s. d. 50 cm.; ma. 25; filter 2 mm. Cu. and 1.25 mm. Al.
13	9/13/38 10/22/38	5,450	2 lateral cervical	8 x 10 cm.	2,450 r. 3,000 l.	28.6 30.7	100- 200	kv. 200; a. s. d. 50 cm.; ma. 25; filter 1.05 mm. Cu. and 1 mm. Al.
14	3/ 3/37 5/11/37	7,000	2 lateral cervical	6 x 8 cm. 8 x 10 cm.	3,500	21.1	100- 200	kv. 200; a. s. d. 50 cm.; filter 2 mm. Cu. and 1.25 mm. Al.
15	5/12/37 6/20/37	7,850	2 lateral cervical	80 cm.²	3,925	6-8	100- 150	A. s. d. 40 cm.; H. V. L. 1.2
16	7/ 8/35 8/ 8/35	7,600	2 lateral cervical	8 x 10 cm.	3,800	5-7	200- 150	A. s. d. 40 cm.; H. V. L. 1.2

Case	Epiglottis	Superior Part of Larynx	Ventricular Band	Vestibule	Vocal Cords	Subglottic Area	Posterior Wall
1	Stratified squamous	Ciliated columnar	Superior portion: ciliated columnar. Inferior: stratified	Ciliated columnar	Stratified squamous	Ciliated columnar	Ciliated columnar

Case	Epiglottis	Superior Part of Larynx	Ventricular Fold	Commissure	Vocal Cords	Anterior Wall	Posterior Wall
1	Stratified squamous	Ciliated columnar	Superior portion: ciliated columnar. Edge: stratified squamous	Ciliated columnar	Stratified squamous	Ciliated columnar	Ciliated columnar
2	Stratified squamous and indifferent stratified, mixed	Stratified squamous	Denuded and stratified squamous	Degenerated stratified columnar and denuded areas	Right: stratified squamous, denuded edge. Left: denuded	Stratified transitional with some signs of degeneration	Stratified squamous, transitional
3	Denuded areas and stratified	Stratified squamous	Stratified squamous	Stratified squamous and membrane, mixed; epithelium around islands of membrane shows degenerative changes and may be lifted by undergrowing membrane			
4	Stratified squamous	Thin stratified squamous and single layer, some degeneration	Thin stratified squamous and denuded areas, mixed	Ciliated columnar; small denued area on left	Upper surface: stratified, squamous. Edge: denuded	Single layer, stratified, squamous and ciliated columnar	Stratified squamous
5	Stratified squamous	Stratified squamous	Superior portion: thin stratified squamous. Edges: denuded	Anterior: nonciliated columnar. Posterior: ciliated columnar	Low squamous epithelium and denuded areas	Stratified squamous	Stratified squamous
6	Stratified squamous and denuded	Stratified squamous decreasing in thickness toward ventricular band	Denuded areas	Ciliated columnar	Denuded areas	Stratified squamous increasing in thickness from above down	
7	Stratified squamous	Stratified squamous	Left: stratified squamous. Right: some cylindric cells with goblet cells	Ciliated columnar, some degeneration	Stratified squamous	Stratified squamous	Stratified squamous
8	Stratified squamous	Stratified squamous	Left: necrotic tissue and membranes Right: stratified squamous epithelium	Stratified squamous	Stratified squamous and ulceration	Stratified squamous	Stratified squamous
9	Stratified squamous	Pseudostratified columnar	Stratified squamous	Ciliated columnar	Stratified squamous	Transitional and ciliated epithelium	Stratified squamous and ciliated columnar
10	Stratified squamous	Indifferent stratified squamous	Stratified squamous	Ciliated columnar	Indifferent and squamous stratified	Stratified squamous	Stratified squamous
11	Stratified squamous	Ciliated columnar	Stratified squamous	Ciliated columnar	Stratified squamous	Transitional and ciliated columnar	Superior: stratified squamous. Inferior: ciliated columnar
12	Stratified squamous and ciliated columnar	Ciliated columnar	Ciliated columnar and free. Edge: stratified squamous	Ciliated columnar	Stratified squamous	Transitional and ciliated columnar	Ciliated columnar
13	Stratified squamous and transitional with islands of ciliated columnar			Stratified squamous and ciliated columnar	Stratified squamous	Transitional and ciliated columnar	Similar to subglottic area
14	Stratified squamous	Stratified squamous	Stratified squamous	Left: ciliated columnar. Right: islands of ciliated columnar and stratified squamous	Stratified squamous	Stratified squamous, transitional and ciliated columnar	
15	Stratified squamous	Stratified squamous	Squamous epithelium	Thin squamous epithelium	Stratified squamous. Right: denuded	Stratified squamous	Stratified squamous
16	Stratified squamous	Stratified squamous	Stratified squamous	Ciliated columnar	Right: ulcerated. Left: stratified squamous	Right: ulcerated, stratified squamous. Left: stratified indifferent	Stratified indifferent

the columnar epithelium of the interior of the larynx and less so in the ventricle (fig. 1). In only 1 larynx was there complete replacement of the ciliated columnar epithelium of the ventricle by stratified squamous epithelium (case 8). Only 2 (cases 1 and 6) showed no changes of the ciliated columnar epithelium. The remaining ones showed small islands of degenerative change, nonciliated elements or membrane formation. Changes of the columnar epithelium cannot be considered as specific sequels of irradiation, for they are also found in the course of normal embryonic development and in many conditions in which chronic laryngeal irritations are present.

In agreement with Patzelt,[4] we consider these processes not as true metaplasia but as reconstruction of epithelium following changes in the differentiation of the deeper undifferentiated basal cells, changes which here may be caused by roentgen radiation. The changes are always from ciliated to squamous epithelium and never in the reverse direction. Necrosis is rarely found in ciliated columnar cells but is present in the undifferentiated basal cells with a resultant separation of the other cells from the basal membrane.

Sometimes (case 7) one finds under the basal cells circumscribed areas of serum and fibrin, resulting in a lifting of the epithelium (fig. 2). In areas where epithelium undergoes necrosis and sloughing occurs, a fibrinous membrane, consisting of a fibrinous network containing a varying number of leukocytes and round cells, is eventually formed. In 3 of the larynxes (cases 2, 5 and 6) the cellular infiltration of the membrane was less marked than in those with fibrinous membrane resulting from inflammatory reactions of an infectional type. In 1 larynx, examined twenty days after the onset of radiation (case 3), there was the usual picture of an intensive leukocytic infiltration of the membranes.

Peculiar pictures are sometimes seen in the areas where the columnar epithelium and the squamous epithelium join. In 1 larynx there were degenerative changes in the ciliated epithelium for a short distance and under this degenerated area squamous epithelium had grown in tongue-like projections and caused detachment of the ciliated epithelium (fig. 3). This is apparently a squamous epithelial replacement of the degenerating columnar epithelium.

Necrosis of the epithelium is most frequently found in the aryepiglottic fold, on the vocal cord, on the ventricular band, on the posterior laryngeal wall and on the upper part of the laryngeal surface of the epiglottis—areas which are, as a rule, covered with stratified squamous

4. Patzelt, V.: Ueber die menschliche Epiglottis und die Entwicklung des Epithels in den Nachbargebieten, Ztschr. f. Anat. u. Entwcklngsgesch. **70**:1. 1923.

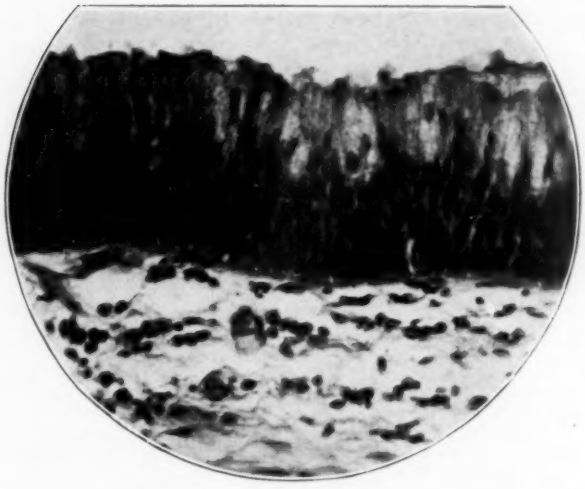

Fig. 1.—Transformation of pseudostratified columnar epithelium to stratified squamous epithelium.

Fig. 2.—Subepithelial exudation of fibrin and serum.

epithelium. The ciliated epithelium of the ventricle remained more or less intact. The regularity of this localization of the necrosis may be due to greater radiosensitivity of the squamous epithelium. Irritation may, however, be a factor, inasmuch as carcinoma is frequently preceded by a chronic inflammatory irritation of the mucosa, or this irritation may be caused by the presence of cancer itself in the interior of the larynx, in both of which circumstances there might be a substitution of squamous epithelium for columnar epithelium.

Preceding the formation of a surface fibrinous membrane there is always necrosis of the surface epithelium; there may, however, be areas which lie between two denuded areas, and then the fibrin which surrounds or adjoins the intact areas may grow across the intact epithelium

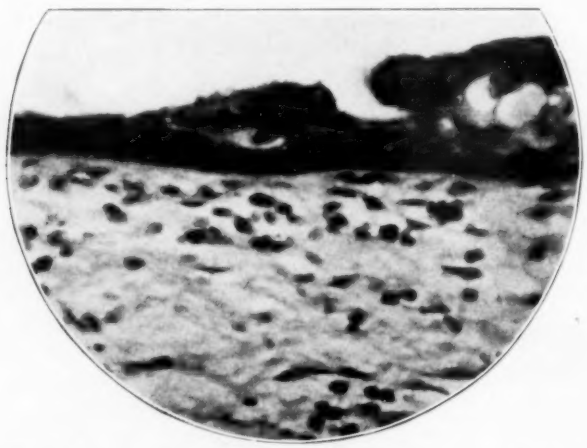

Fig. 3.—Substitution of pseudostratified columnar epithelium by stratified squamous epithelium.

and cause a secondary degenerative change (fig. 4 *A*). This was noted in 3 of the 8 larynxes. In 2 the epithelium at the margin of the denuded areas showed degenerative changes indicating that necrosis was still in progress. Intensive cellular infiltration of the fibrinous membrane, with predominance of leukocytes, was found in 1 larynx (case 3), which was examined soon after the onset of radiation. In the later stages the number of cells diminishes. Subsequently the membrane is sloughed off and reepithelization progresses rapidly from the margin (fig. 4 *B*). This was noted in 2 larynxes. Eventually all of the denuded areas, even those originally covered with columnar epithelium, are covered with stratified squamous epithelium. This suggests a tendency to rapid reepithelization and agrees with clinical experience, i.e., the laryngeal mucosa is covered with intact epithelium after the loss of a fibrin coating.

The acute reaction of the epithelium of the oral cavity to radiation has been studied by Borak [5] (1934), who found two different reactions to radiation: (1) epitheliolysis, a necrosis of the epithelium with inflam-

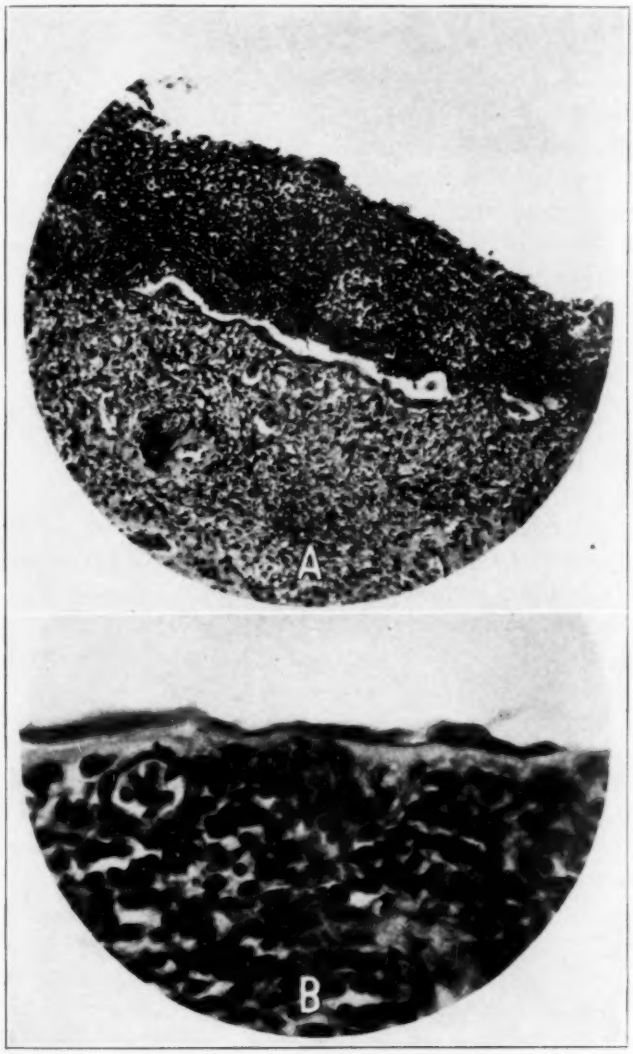

Fig. 4.—*A*, fibrinous membrane and *B*, reepithelization.

5. Borak, J.: (*a*) Ueber radiogene Hyperkeratosen der Schleimhaut, Virchows Arch. f. path. Anat. **294**:304, 1934; (*b*) Ueber die epidermiolytische Bestrahlungsreaktion, Fortschr. a. d. Geb. d. Röntgenstrahlen **45**:397, 1932; (*c*) Die roentgentherapeutische Behandlung der oropharyngolaryngealen Carcinome, Monatschr. f. Ohrenh. **69**:1108, 1935.

matory processes, and (2) epitheliokeratosis, a degenerative process without inflammatory reactions. The findings in the cases presented show that the changes in the larynx correspond with the epitheliolytic type of Borak, insofar as the squamous epithelium is concerned. In the ciliated columnar epithelium the transformation into stratified squamous epithelium is somewhat similar to the epitheliokeratotic type, whereas the degenerative changes occurring in the more affected larynxes are similar to epitheliolysis.

The mucosal changes vary in intensity in the different larynxes but are uniform in character. The changes are as a rule limited to the superficial layers of the mucosa and decrease in intensity as the depth of the mucosa increases. The earlier changes may be said to be simple hyperemia and beginning cellular emigration. Later reactions in the vascular, connective tissue, glandular and cellular elements occur at the same time and are interrelated. There is continued increase in the fibrillar element of the mucosal connective tissue. This is most marked in the areas covered with fibrin and may be accompanied by exudation of fibrinous masses within the mucosa itself. These fibrinous masses may or may not be in connection with the surface fibrin. Similar fibrin masses are sometimes found in areas covered with epithelium.

There is during this period at first a more or less intense diffuse inflammatory infiltration with a relative diminution of polymorphonuclear neutrophils, which is later superseded by marked decrease in the amount of subepithelial lymphocytic infiltration and decided increase in plasma cells. In 2 cases the fibrous tissue had begun to undergo hyaline degeneration. In 3 the fibrous reaction extended deep enough to cause secondary fibrosis of the muscular tissues.

The vascular reaction begins as simple hyperemia and cellular emigration with damage to the capillaries and precapillaries in the form of dilatation, formation of thrombi and exudation of serum and fibrin (noted in 7 of 8 larynxes). At times there may be small hemorrhages into the tissues, and in 2 larynxes there was marked enlargement of the lymph spaces. Later there may be endothelial hyperplasia and occlusion of the smaller vessels (7 of 8 larynxes). Similar changes are less frequently noted in the larger vessels. The vessels are affected unequally in different areas, and different vessels are affected unequally, with the more marked changes occurring in the smaller vessels and particularly the veins, such changes being found in the larger vessels in only 5 instances.

At the time that reepithelization begins the superficial layers of the mucosa undergo fibrotic changes, i. e., degeneration of many of the fibroblasts and thickening of the connective tissue fibrils. At the same time, in some of the medium-sized vessels, which were in the beginning, with few exceptions, intact, there are proliferation of the endothelium

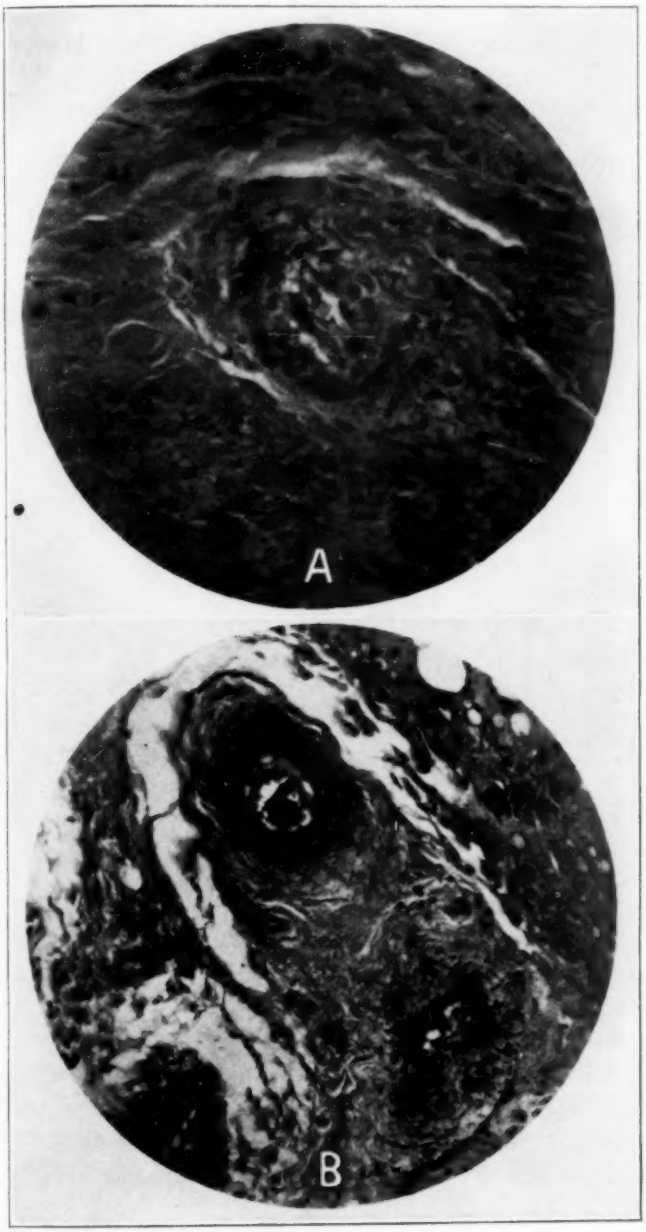

Fig. 5.—*A*, early vascular change and *B*, late vascular change.

with narrowing of the lumen, cellular infiltration of the media leading to partial destruction of the membrana elastica interna, splitting of the elastic lamellae of the media and eventually fibrosis of the media, and cellular infiltration and fibrosis of the adventitia. Occasionally swelling and hyaline degeneration of the muscle fibers of the media are observed (fig. 5).

The glandular picture, similar in all instances, may in a single area include beginning degeneration, complete atrophy and a normal appearance of glandular lobules. The most superficial glands are the most

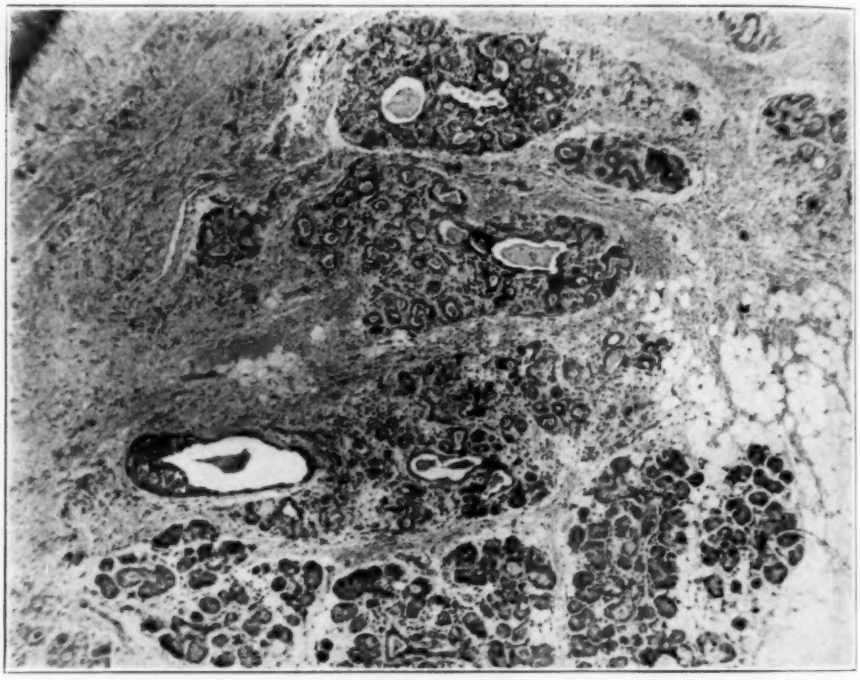

Fig. 6.—Glandular changes.

affected. The glandular ducts are usually lined for a short distance from their orifice by the same epithelium that lines the area in which they lie. In 6 of the larynxes examined there was dilatation of some of the glandular ducts, while in 2 there was definite cystic formation. The degeneration of the glandular portions is marked by swelling of the nuclei and protoplasm, which may accompany round cell and plasma cell infiltration of the glandular lobules. The glandular changes also seem to parallel in some degree the intensity of the fibrotic changes in the mucosa, the glands lying in the areas of greatest fibrotic changes showing the most marked atrophy while those which lie in the relatively normal areas show fewer lobules affected (fig. 6).

In general it may be said that the mucosa shows the greatest change in the areas of epithelial necrosis, a lesser change in the areas of intact epithelium and the least change in the ventricles; the last fact is difficult to explain satisfactorily, though it may be due to the peculiar anatomic position of the ventricles, relatively far from the laryngeal lumen and near the interior surface of the thyroid cartilage.

Fig. 7.—Perichondritis of thyroid cartilage.

The remaining laryngeal tissues (cartilages, muscles and others) show no radiation changes in the acute stage. In 1 larynx (case 5), however, there was necrosis of the cartilage. This was considered to have been present before the start of roentgen treatment. Histologic examination of this larynx showed internal and external perichondritis of the right ala of the thyroid cartilage. No histologic peculiarity was noted, but inasmuch as prelaryngeal swelling was described clinically previous to the start of radiation therapy, and since the picture was one of pyogenic infection, the perichondritis is considered to have been a

so-called secondary perichondritis originating after ulceration of the tumor of the left cord, perhaps a consequence of the first biopsy (fig. 7).

HISTOLOGIC OBSERVATIONS ON LARYNXES EXAMINED MORE THAN THREE AND ONE-HALF MONTHS AFTER THE TERMINATION OF RADIATION THERAPY

A summary of the clinical records of the patients from whom the second group of larynxes were taken (cases 9 to 16) is found in table 4.

Acute reactions of the laryngeal mucosa to roentgen therapy are rather charcteristic and well defined, but it is difficult to differentiate the changes observed months or years after the termination of radiation therapy and considered to constitute the picture of the late stage of reaction to radiation from the changes which are due to extended periods of irritation. It is important to remember that chronic irritative changes of the mucosa of the larynx belong to the characteristic clinical picture of cancer of the larynx. These changes may be manifestations of the chronic irritation from the long-standing catarrhal conditions, excessive vocal strain or excessive use of alcohol and of tobacco that preceded the development of the cancer, or they may be caused by the presence of cancer in the interior of the larynx. One must, therefore, attempt to separate such alterations from those which are due to irradiation of the larynx.

Larynxes affected by simple chronic catarrhal conditions, not complicated by carcinoma, show grossly diffuse or irregular thickening of the mucosa. Histologically there are islet-shaped or more extensive transformations of the ciliated epithelium to squamous epithelium, sometimes pachydermas with cornification and chronic inflammatory cell infiltration of the superficial layers of the mucosa and later increase of fibrillar connective tissue. These changes are most marked in the vocal cords and ventricular bands and on the posterior laryngeal wall. During the earlier stages there is a tendency to edema in the loose subepithelial layer of the vocal cord (Reinke's space) and in the submucosa of the subglottic region. Similar changes are observed after irradiation of the larynx (fig. 8), but in addition there are obliterative vascular changes and edema of atypical localization.

The mucosa in these 8 larynxes, all of which were examined three and one-half months or more after the onset of radiation therapy, showed the following changes:

There were few changes in the epithelium following reepithelization of the denuded areas. The transformation of the ciliated epithelium into squamous epithelium had continued and advanced until, in 2 larynxes, there was an almost complete lining of stratified squamous epithelium. As a rule, however, the ciliated epithelium in the ventricles remained pre-

TABLE 4.—*Clinical Data on Last Eight Cases (Sections from Larynx Examined Over Three and One-Half Months After Irradiation)*

Case	Sex	Age, Years	Clinical Course	Observations at Autopsy
9	M	70	Hoarseness 1 mo.; radiation, 6/27/29 to 7/27/29 and 9/16/29 to 10/19/29; regression of tumor during first course; disappearance during second course; readmission 6/23/30 with tumor in left piriform sinus; death 7/16/30	Generalized arteriosclerosis; lobular pneumonia with abscess formation in dependent parts; metastases to left tracheobronchial lymph nodes, lungs, parietal pleura and larynx (see table 5)
10	M	59	Pain in right side of neck 10 mo.; radiation, 3/20/35 to 4/17/35 and 4/24/35 to 4/26/35; membrane formation beginning 15 days after start of radiation, involving posterior pharyngeal wall, piriform sinuses and entrance of larynx; gradual disappearance of membrane and tumor; rest of tumor at base of tongue; 540 mg. hr. of radium with disappearance of rest, 6/19/35; hemorrhage 8/8/35; death 8/9/35	Ulceration of vallecula and base of tongue; tumor infiltrating base of tongue and epiglottis; right aryepiglottic fold edematous
11	M	58	Dysphagia 2 mo.; radiation, 9/3/37 to 10/9/37; membrane formation in 3½ weeks over surface of tumor, then ventricular bands and right piriform sinus; regression of tumor and glands; 4 weeks after radiation, local recurrence with ulceration; death from hemorrhage 2/8/38	Blood in bronchial tree; metastases to both lower lobes subpleurally; cavity in region of right hyothyroid membrane, filled with necrotic tissue and blood clots; erosion of internal jugular vein; edema of right vallecula, right arytenoid and aryepiglottic fold; metastasis to right cervical glands and tracheobronchial glands
12	M	69	Hoarseness 1 yr.; radiation, 4/27/36 to 5/21/36; no membrane formation; premature discontinuance of radiation; 10/16/36 amputation of right leg for diabetic gangrene of toe; death 10/24/36	Generalized advanced arteriosclerosis and lobular pneumonia; ulcer below right vocal cord; slight edema of aryepiglottic folds and laryngeal mucosa, especially over vocal cords
13	M	72	Dysphagia and hoarseness 3 weeks; radiation, 9/13/38 to 10/22/38; rapid regression of tumor; membrane formation over laryngeal structure and hypopharynx beginning 20 days after start of radiation and persisting 2½ weeks; removal to institution for patients with mental disorder necessitated by psychosis; death 3/9/39	General arteriosclerosis and extensive softening of left basal ganglions; terminal pneumonic congestion; vocal cords and ventricular bands thickened
14	M	48	Hoarseness 2 yr.; radiation, 3/3/37 to 5/11/37; gradual decrease of tumor; after 1 mo. questionable mass on right cord; 8/25/37 signs of myocardial infarction; death 10/2/37	Coronary sclerosis with thrombosis of anterior descending left coronary artery; old and recent infarcts of left ventricle with aneurysm formation; entire larynx edematous; no tumor seen
15	M	55	Hoarseness 4 mo.; radiation, 5/12/36 to 6/20/36; regression of tumor; membrane formation over superior portion of larynx and hypopharynx in 3 weeks; 3 mo. later recurrence in original site; 1/5/37 readmission with extensive recurrence; laryngectomy 1/14/37; death 1/30/37	Lobular pneumonic foci with suppuration in both lungs; larynx showing ulceration of right side involving anterior commissure, median line posterior, ventricular band and vocal cord and portion of epiglottis
16	M	52	Intermittent hoarseness 2 yr.; radiation, 7/7/35 to 8/8/35; complete regression of tumor; membrance formation on right aryepiglottic fold 18 days after institution of radiation; 1/3/36 readmission with recurrence on right cord; laryngectomy 1/13/36	Resected larynx: oval-shaped ulcer on right, involving vocal cord and subglottic area; edema of right vocal cord

served. In those cases in which the tumor was extralaryngeal, changes are considered to be due entirely to radiation rather than to irritation. Here, despite large doses of roentgen rays, ciliated epithelium was present in the ventricles and in some scattered areas of the interior of the larynx. The ventricles were lined with ciliated epithelium in all but 2 larynxes, and in one of these the tumor-free half of the larynx

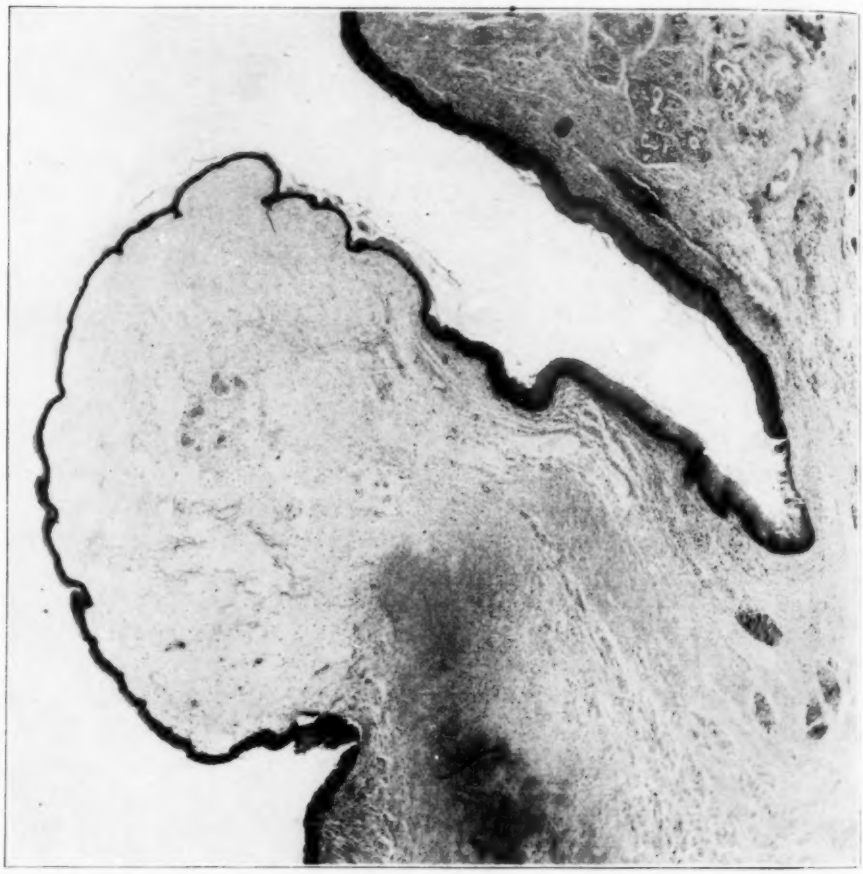

Fig. 8.—Edema of Reinke's space.

showed no transformation of the ciliated epithelium in the ventricle, while in the other there was only partial substitution of the ciliated epithelium of the ventricle. Generally there was no abnormality of the stratified squamous epithelium, but in 1 larynx there were eleidin granules in the epithelium covering the ventricular band areas, and 2 larynxes had a slight increase in the thickness of the epithelium in their superior portions.

The reaction of the superficial layers of the mucosa, the atrophy of the glands and the vascular changes noted in the acute stages, had continued and there was more or less marked fibrosis, which in some cases extended into the deeper layers. Fibrosis was usually most marked in the subglottic region, the posterior wall, the ventricular band and the vocal cord, while it was least marked in the ventricle. The intensity of the fibrosis varied in the different cases, being usually only moderate. One larynx showed hyalinization of the connective tissue (fig. 9). Subepithelial infiltration of round cells was decreased, but in 1 larynx there were newly formed areas of round cell accumulations which resembled lymph follicles.

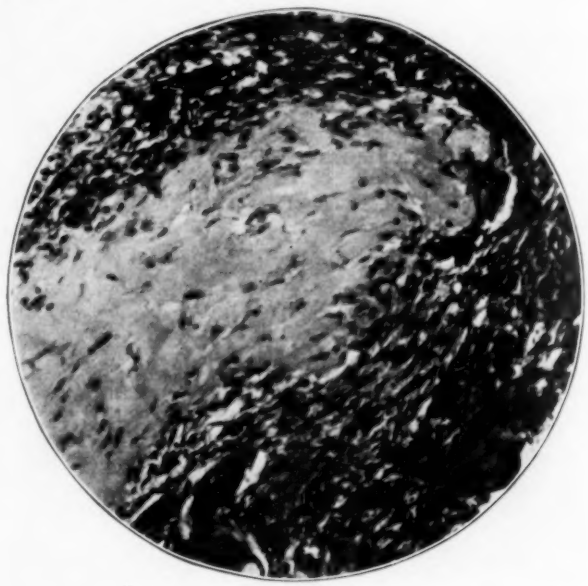

Fig. 9.—Progressive hyalinization of connective tissue.

The obliterative changes in the small and medium-sized vessels had continued and were more marked in some of the smaller submucosal vessels, while the larger vessels were as a rule, unaltered. Some of the larger vessels showed intimal proliferations, but as all the larynxes were those of old people this process must not necessarily be regarded as a sequela of radiation treatment. Relatively few vessels were affected in any one area, and the changes were always more frequent in veins than in arteries.

In the acute stages exudation of fluid into the superficial layers of the mucosa was noted, and later, at about the time of development of the fibrosis, edema was found in certain locations. Edema was also found in the later stages; usually it was in the following situations: the loose

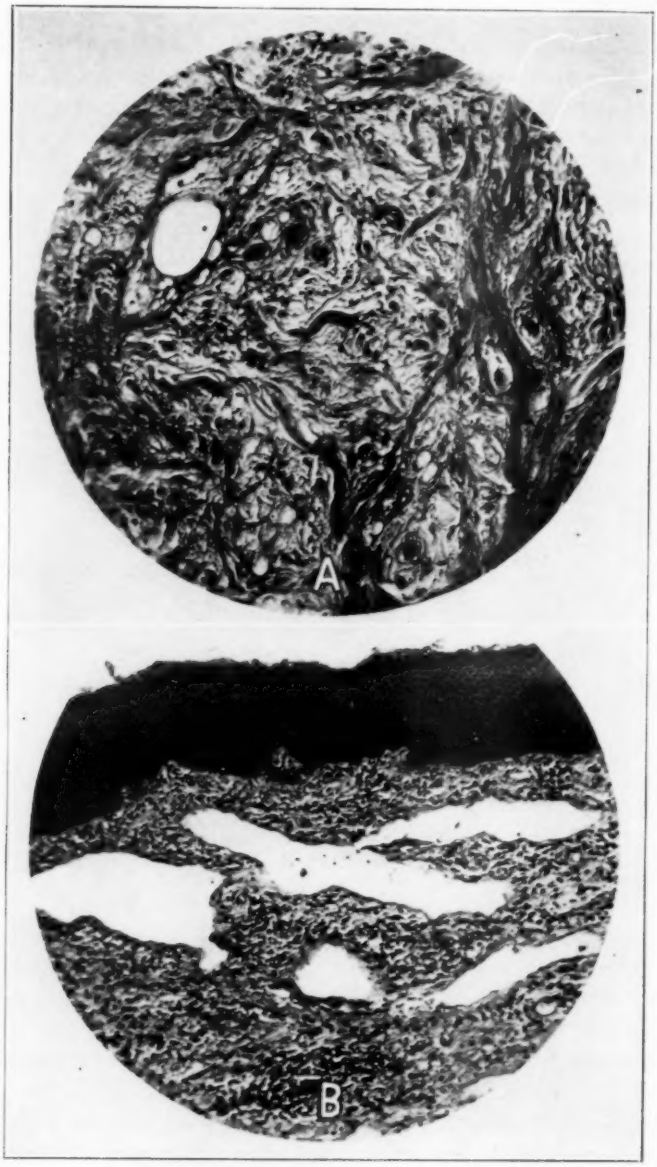

Fig. 10.—*A*, fibrous organization of edema of the subglottic region and *B*, dilatation of lymph vessels of the subglottic region.

subepithelial tissue of the vocal cord (Reinke's space) (fig. 8), where it is also found in chronic laryngitis; in the loose connective tissue of the subglottic spaces; less frequently in the tissue of the laryngeal surface of the epiglottis, which with the exception of the petiolus is otherwise free of edema; sometimes in the neighborhood of the thyroid or the cricoid cartilage. Fibrous organization of the edematous accumulation occasionally occurs (fig. 10 *A*). The development of the edema is apparently aided by disturbances of circulation due to the vascular changes, but a relationship could not be discovered.

In 3 larynxes there was marked dilatation of the subepithelial lymph vessels, which may also be regarded as a manifestation of circulatory disturbances (fig. 10 *B*).

In areas adjacent to the fibrotic changes the muscle fibers were sometimes involved and showed secondary atrophy, but none of the larynxes showed primary damage of the muscle bundles caused by radiation.

The bones, cartilages and perichondrium likewise showed no changes due to irradiation of these tissues. The ossified portions of the cartilage looked normal except for almost complete lack of hemopoietic marrow, a deficiency which might be caused by radiation.

EFFECTS OF RADIATION ON TUMOR TISSUES

This is neither the time nor the place to discuss the advantages and disadvantages of roentgen therapy, or the use and misuse of the Coutard principle in particular. But it should be recognized that there are many factors—constitutional, anatomic and therapeutic—which must be considered in arriving at the radiosensitivity of any single growth. Here, however, the purpose is not to condone or condemn any therapeutic approach, whether surgical or roentgenologic, but is rather to indicate the changes which have taken place in the laryngeal and the tumor tissue after radiation has been applied, in keeping, more or less with the principles of Coutard.

A summary of the results of irradiation is found in table 5.

The classification used in grouping these growths is a modification of that given by Zuppinger,[6] which is essentially the same as that of Duval and Lacassagne:[7]

1. Wholly differentiated epidermoid carcinoma: so-called epidermoid carcinoma. These tumors have their origin in stratified pavement epi-

6. Zuppinger, A.: Archiv und Atlas der normalen und pathologischen Anatomie in typischen Röntgenbildern, Maligne Pharynx- und Larynxtumoren, Leipzig, Georg Thieme, 1931.

7. Duval, R., and Lacassagne, A.: Classification pratique des cancers dérivés des épithéliums cutanés et cutanéo-muqueux, Arch. franç. de path. gén. et expér., 1922, no. 4, p. 1.

TABLE 5.—*Results of Irradiation of Tumors*

Case	Location of Tumor	Total Radiation, (r)	Time Between Final Irradiation and Examination	Biopsy Classification	Clinical Result	Postradiation Therapy; Appearance
1	Left arytenoid, interarytenoid space, aryepiglottic fold and adjacent part of larynx and epiglottis	2,400	4 days	Epidermoid carcinoma, skin type	Laryngectomy performed; mass still present	Similar classification to that of biopsy; lowered mitotic activity; degeneration of some tumor cells; swelling of nuclei; enlargement of nucleolus, chromatolysis, pyknosis, karyorrhexis, homogenization of cytoplasm; stroma unaffected; atypical mitoses
2	Right piriform fossa, right aryepiglottic fold and lateral wall of larynx	5,400 (incomplete)	15 days	Anepidermoid carcinoma	Decrease in size of tumor	Tumor cells greatly degenerated; some cords similar to those seen at biopsy; cells in lymphatics and perineural spaces; mitoses lessened; pyknosis and nuclear and nucleolar swelling marked
3	(*a*) Right and left arytenoid (*b* Vallecula (*c*) Vallecula	9,000 5,400 1,750	20 days	(*a*) Anepidermoid carcinoma (*b*) Semiepidermoid carcinoma (*c*) Epidermoid carcinoma mucosa type	(*a*) Disappearance of tumor in 8 weeks; (*b*) 2½ yr. later ulceration of vallecula, disappearing after radiation; (*c*) ulceration of vallecula; no regression	Tumor in vallecula only; mitoses lowered; otherwise appearance of tumor resembles that seen at last biopsy; marked inflammatory reaction in stroma
4	Right vocal cord, ventricular band and subglottic region	6,700	4 days	Epidermoid carcinoma, skin type	Disappearance of tumor in 4 weeks	Most masses necrotic; few fairly well preserved; some nests showing central cornification at concentric layer arrangement; mitoses almost absent; frequently embedded in thick scar tissue
5	Left cord and subglottic region	8,050	4 days	Epidermoid carcinoma, mucosa type	Disappearance of tumor	Abscess of cartilage; few remainders of tumor; few small, concentrically arranged necrobiotic cells, some surrounded by foreign body giant cells
6	Entire left side of larynx crossing midline anteriorly	7,800	14 days	Epidermoid carcinoma, mucosa type	Regression without complete disappearance	Some cells well preserved, similar to appearance at biopsy, except for lowered mitoses; some cells necrotic, some cornified; stroma rich in cells, infiltrated with round and plasma cells
7	Anterior commissure and anterior ⅓ of right vocal cord	7,650	29 days	Squamous cell carcinoma	Disappearance in 3 weeks	Few small nests of concentrically arranged nuclei-free tumor cells surrounded by foreign body giant cells are all that remain
8	Entire left ventricular band and adjacent mucosa	4,800	51 days	Epidermoid carcinoma, skin type	Regression without disappearance	Cords of tumor cells similar to those seen at biopsy but with marked tendency to keratinization and increased mitoses, some atypical; multinucleated cells; central degeneration of cords with pseudocyst formation; pyknosis and karyorrhexis

9	Left piriform fossa and left aryepiglottic fold	4,800	9 mo.	Squamous cell epithelioma	Decrease in size; recurrenece in 8 mo.	Semiepidermoid carcinoma; tumor cells in large masses with scattered stroma of small strands of connective tissue; mitoses common; no intercellular bridges but whirlpool arrangement with central keratinization; cells large with nuclei varying in size, shape and staining reaction
10	Right vallecula, invading right tonsil and right laryngeal wall and base of tongue	7,700 plus 500 submental 540 mg. hr. radium	3½ mo.	Epidermoid carcinoma, mucosa type	Decrease in size; 3½ mo. later, hemorrhage from ulcer in vallecula	Tumor composed of cords; appearance of some cells similar to that seen at biopsy; some central necrosis and formation of detritus-filled cavities; some parakeratotic cornification in interior; mitoses similar to those at biopsy; stroma cell poor and thick; some foreign body giant cells surrounding necrotic nests
11	Right piriform sinus and right ventricular band	8,050	4 mo.	Transitional cell carcinoma	Disappearance of tumor in 8 weeks; 4 weeks later, ulcer at site of tumor; death from hemorrhage from ulcer	No carcinomatous tissue in larynx; abscess in piriform fossa; no carcinomatous tissue
12	Middle ⅓ of right cord	4,400 (incomplete)	6 mo.	Epidermoid carcinoma, mucosa type	Gradual disappearance of tumor	Some areas of same character as biopsy specimen; mitoses almost absent; some degeneration—polynucleated cells, bizarre cells, pyknosis, karyorrhexis, vascular degeneration and hyalinization of cytoplasm; rich in thin fibrils and infiltrated with lymphocytes and plasma cells; more fibrous at periphery of growth
13	Right arytenoid, ventricular band, aryepiglottic fold and base of epiglottis	5,450	4½ mo.	Semiepidermoid carcinoma	Complete regression of tumor	No carcinoma tissue
14	Right arytenoid, ventricular band and cord; crossing anterior commissure	7,000	5 mo.	Epidermoid carcinoma, skin type	Decrease in tumor 1 mo. after irradiation; questionable mass on right cord	Few remainders of necrotic tumor cells or lamellated horny masses
15	Right cord crossing anterior commissure	7,850	7 mo.	Epidermoid carcinoma mucosa type	Rapid complete disappearance, 4 weeks; local recurrence 5 mo.	Cords of tumor cells similar to those noted at biopsy; clinical regression of tumor without complete histologic destruction of tumor cells
16	Right subglottic growth crossing midline	7,000	2½ yr.	Epidermoid carcinoma, mucosa type	Regression of tumor; local recurrence 2½ yr. after radiation; laryngectomy	Tumor showing higher differentiation than at biopsy; many areas of parakeratotic keratinization; stroma infiltrated

thelium and retain more or less the structure or developmental stages seen in the epidermis of the skin or mucous membranes (skin type and mucous membrane type).

2. Moderately differentiated epidermoid carcinoma: so-called semi-epidermoid carcinoma. These tumors either show moderate epidermoid differentiation, which may remain at an early level, i.e., carcinoma with moderate differentiation, or contain totally undifferentiated parts along with the parts in which there is moderate epidermal differentiation. There may be cells concentrically arranged, forming stratification balls in the inner portion of the tumor cord.

3. Anepidermoid carcinoma: These tumors show no differentiation into normal epidermis.

The tumors are also differentiated according to the manner of growth: (1) exophytic carcinoma which grows toward the outside; (2) infiltrating carcinoma, which grows into the tissue, and (3) fungoid carcinoma, which grows in both directions.

The 16 tumors reported on were all from men of advanced years. There were 12 tumors of the larynx, 3 of the piriform fossa and 1 of the vallecula. Anatomically there were 9 intrinsic and 3 extrinsic cancers of the larynx. In all instances of intrinsic cancer except 1 (case 15), in which together with the local recurrence, metastases in the cervical glands developed, there were no metastases. In the 3 instances of extrinsic growth (cases 1, 3 and 13) metastases occurred, though these were not always manifest at the time of onset of radiation therapy (cases 3 and 13) or operation (case 1). In case 1 metastases became manifest in the cervical glands after nine months, and in case 3, in the vallecula after two and one-half years. The site of origin could be determined in 9 cases (3, 4, 5, 7, 8, 12, 13, 15 and 16). Two tumors were supraglottic; 1 was from the left ventricular fold (case 8); 1 arose from the anterior part of the right arytenoid cartilage (case 3); 5 originated from the vocal cords (cases 4, 5, 7, 12 and 15), and 1 was subglottic (case 16). Only 1 tumor of the cord (case 12) was confined to the cord, 1 (case 5) having encroached on the adjacent parts of the subglottic region, 2 (cases 7 and 15) having infiltrated the anterior commissure and 1 (case 4) having infiltrated more extensive parts of one half of the larynx and, in addition, crossed the midline anteriorly.

All of the tumors except 1 (case 1) were diagnosed as fungoid carcinoma. There were no purely exophytic tumors spreading over the surface of the mucosa in this series. Classified as to the portion of the larynx occupied—anterior, middle or posterior—there were 2 anterior (cases 7 and 15), 2 posterior (cases 3 and 13) and 7 middle tumors, while the remainder did not lend themselves to this classification.

A reaction in the mucous membrane of the larynx, a so-called radio-epithelitis with formation of fibrinous pseudomembranes, was observed in the majority of the larynxes which received more than 5,000 roentgens, but it varied considerably as to time of appearance and duration. A relation between the intensity of this reaction and the radiosensitivity of the tumor could not be found. The theory of E. G. Mayer [8] and Wessely [9] that the duration and the intensity of the reaction of the mucosa depend not only on extrinsic factors, such as total dose and division of radiation, but also, largely, on the individual reaction of the patient, is supported by these observations.

It is known that radiation causes degenerative changes and necrosis of varying degree in tumor cells, but it is also known that spontaneous degenerative changes are common in epithelial tumors; as these changes vary and do not affect all parts of the neoplasm equally, it is sometimes difficult to decide whether they are spontaneous or secondary to irradiation of the tumor. The changes in the tumors concerned here were rather uniform and agreed essentially with those described by other authors as occurring in tumors receiving fractional doses of radiation. So the possibility that they were the result of irradiation of the tumors seems very likely. The status of the cases in which there was complete destruction of the tumor, with complete disappearance of all tumor cells, is no problem. In 1 instance, however, there was a transformation of some of the groups of the tumor cells into hyaline or horny masses, surrounded by granulation tissue and foreign body giant cells, which might be considered as representing late cellular evolution under the influence of radiation, similar to the evolution undergone by cells of cornifying stratified squamous epithelium in normal conditions.

Several authors, Aschoff, Krönig and Gauss [10] (1913) and Hamperl and Schwarz [11] (1927), have observed and reported the ripening of tumors under the influence of radiation. This may explain the observations in 2 cases of epidermoid carcinoma of the mucosa type, in which the neoplasm showed in the anatomic specimen a higher degree of differentiation than it did in the biopsy specimen. In case 3 the primary

8. Mayer, E. G.: (*a*) Die Schwierigkeiten bei der Durchtührung der Bestrahlung nach der Coutardschen Methode, Monatschr. f. Ohrenh. **68**:1146, 1934; (*b*) Probleme der Röntgentherapie des Carcinoms, Strahlentherapie **66**:233, 1939.

9. Wessely, E.: Zur fraktionierten und protrahiert fraktionierten Langzeitbestrahlung (Coutard) der malignen Tumoren des Larynx und Pharynx. Klinische Beobachtungen, Monatschr. f. Ohrenh. **68**:1351, 1934.

10. Aschoff, Krönig, and Gauss: Zur Frage der Beeinflussbarkeit tiefliegender Krebse durch strahlende Energie, München. med. Wchnschr. **60**:337 and 413, 1913.

11. Hamperl, H., and Schwarz, G.: Zur genaueren Kenntnis der Roentgenwirkung auf Krebsgeschwülste, Strahlentherapie **24**:607, 1927.

tumor situated in the larynx was classified as anepidermoid carcinoma, but the classification based on subsequent biopsy of specimens of a metastasis in the vallecula was semiepidermoid carcinoma and epidermoid carcinoma of the mucosa type, the classification of the anatomic specimen being similar to that made at the last biopsy. As Körbl [12] (1912) noted in cases of irradiation of basal cell carcinoma a higher differentiation in the metastatic than in the primary growth, it would seem that the steadily progressive differentiation of the metastatic growth in this instance was also caused by radiation. Swelling of the nuclei with enlargement of the nucleoli followed by pyknosis or chromatolysis, as well as pyknosis and swelling of the protoplasm with vacuolation or hyaline degeneration, is considered a regular consequence of irradiation of a tumor. These changes were frequently found in tumors examined shortly after the use of radiation (cases 1, 2, 3 and 6), less often in those which had been irradiated for a longer time. Besides these more or less characteristic changes, other signs of degeneration, such as pyknosis, karyorrhexis and karyolysis, were present. A diminished number or an absence of mitoses is a common finding.

Alberti and Politzer [13] have shown experimentally, and Ewing [14] has stressed, that after irradiation of a tumor there is always a period during which mitoses are reduced in number and that when they again become numerous they are abnormal in form and probably also abortive. The period of arrestive growth varies from days to months and is dependent on the character of the tumor and the dose of radiation received. Among the 12 cases in which carcinomatous tissue was present at the time of examination a comparison of mitoses was possible in 11. In 5 cases the tumors were examined within fifteen days after the termination of the treatment with radiation, and diminution or absence of mitoses was found in 4; in 1 some areas of mitoses were present. Six were examined from ten and one-half months to two and one-half years after irradiation; 1 showed a marked diminution, while the others showed either no change or a slight increase in the number of mitotic figures and frequent typical mitoses and multinucleated cells. These findings may have been caused by radiation, but such changes are also found in nonirradiated tumors and therefore cannot be considered as the effect of radiation alone when they are found in a specimen after application of radiation but were not found in the relatively small area of the autopsy specimen.

12. Körbl, H.: Die Röntgenbehandlung der Hautcarcinome, speciell des Basalzellenkrebses; sein histologisches Verhalten vor und nach der Bestrahlung, Arch. f. klin. Chir. **97**:752, 1912.

13. Alberti, W., and Politzer, G.: Experimentalbiologische Vorstudien zur Krebstherapie, Fortscher. a. d. Geb. d. Röntgenstrahlen **32**:56, 1924.

14. Ewing, J.: Tissue Reaction to Radiation, Am. J. Roentgenol. **15**:93, 1926.

Changes in the stroma of irradiated tumor, interesting from both the practical and the theoretic point of view, are difficult to evaluate, for certain of them are found in nonirradiated tumors and are considered either morphologic manifestations of the normal defense mechanisms of the body against invading tumor cells or reactions to spontaneous degenerative changes within the tumor. It has been stated that one of the most significant features of cancers is their power of exciting growth of new cellular tissue. Often the cellular tissue is replaced by, preceded by or associated with, lymphoid infiltration, and commonly the process terminates in fibrosis; such changes have to be distinguished from those due solely to radiation, which either acts on the stroma or influences the stroma secondarily by a primary destruction of the carcinomatous tissue, a process which may also terminate in fibrosis.

An early fibrotic transformation of the stroma may occur before complete destruction of the tumor cells (abortive fibrosis, Ewing [14]), and may decrease and eventually prevent the cure of the tumor by radiation, apparently through the protection of the cells afforded by the dense fibrous tissue. In the specimens reported on here the stroma of the tumors shows essentially the same changes as the tumor-free laryngeal muscosa, but because the stroma is infiltrated not only by the roentgen rays but by tumor cells as well, the changes were always more marked. The appearance of numerous neutrophilic and sometimes of eosinophilic leukocytes in the center of the decomposing tumor cells, the emigration of leukocytes in many parts of the tumor cords and the decrease of round cell infiltration around the tumor masses may be considered indirect sequelae of the use of radiation. In addition, surrounding granulation tissue and foreign body giant cell, or cholesterol gaps in the area of necrotic cancer, or horny masses, may be considered as radiation effects. In those areas where the carcinomatous tissue had been completely destroyed, the vascular and fibrotic type of changes were the most marked. The fibrotic tissue at times showed signs of hyalinization. The extent of the changes apparently depends on the extent of the carcinoma. The fibrosis frequently extended to the musculature and led to secondary fibrotic changes of the muscle bundles. There was generally a certain parallelism between the intensity of the degeneration of the tumor and the intensity of the fibrotic changes. Intact carcinoma cells were, as a rule, surrounded by a moderately fibrotic mucosa, there being only 1 case (4) in which fairly well preserved carcinomatous tissues were found in markedly fibrotic tissue (resembling abortive fibrosis) (fig. 11).

In 10 of 16 tumors—or if 1 (case 1) which had only a short course of preoperative irradiation and 2 (cases 2 and 12) the irradiation of which was interrupted prematurely are excluded—in 10 of 13 tumors a primary

regression was accomplished. Among the 10 laryngeal tumors, primary regression occurred in 8.

Insofar as definite conclusions can be drawn from examination of numerous slides from different sections of the larynx, it may be stated that an anatomic cure has occurred in only 4 cases (5, 7, 13 and 14). The lesions in these cases were: (1) an isolated squamous cell carcinoma of the vocal cord, the latter showing unimpaired motility (case 7); (2) an epidermoid carcinoma of the skin type, which infiltrated the cord and the ventricular band, crossed the midline and caused a moderate decrease in

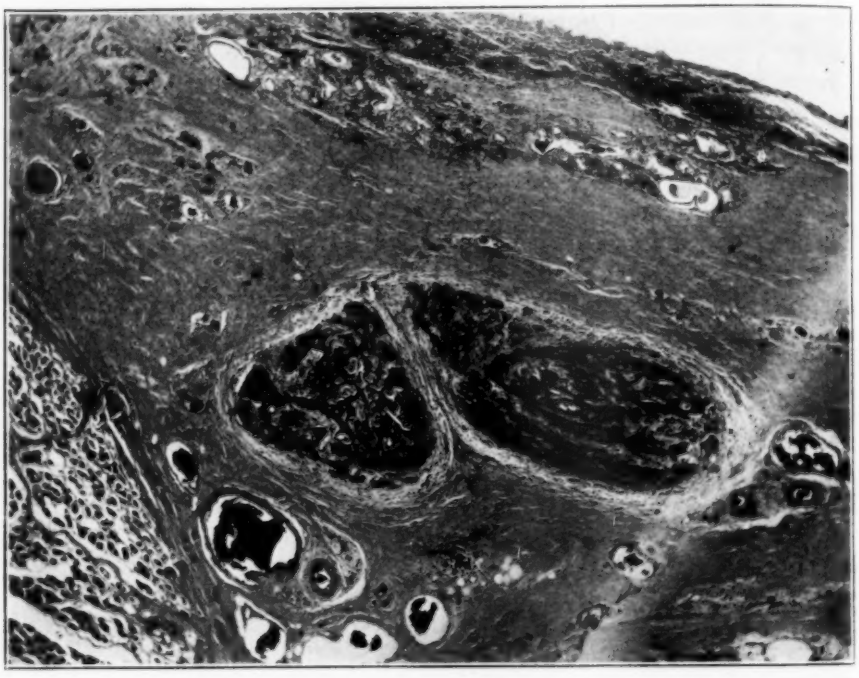

Fig. 11.—Abortive fibrosis.

the motility of the cord (case 14); (3) an epidermoid carcinoma of the mucosa type, involving the vocal cord and encroaching on the subglottic space, but associated with almost normal motility of the vocal cord, and complicated by perichondritis of the thyroid cartilage, apparently originating from an infection following the removal of tissue for biopsy (case 5), and (4) a semiepidermoid carcinoma originating in the region of the right arytenoid cartilage with metastases in the cervical gland (case 13). Three of these were superficially situated, easily accessible tumors, which did not infiltrate either the muscle or the cartilage. All of them except 1 received relatively high doses of radiation (7,650 roentgens in case 7;

7,000 roentgens in case 14; 8,050 roentgens in case 5, and 5,450 roentgens in case 13), and though at least 2 of the tumors were well differentiated and relatively radioresistant, all disappeared under irradiation. This would seem to confirm the opinion expressed by Coutard, that in cases of laryngeal tumor the localization and the type of growth are of greater importance than the histologic structure.

A few other points seem worth mentioning. The growth in case 14, a well differentiated epidermoid carcinoma of the skin type, disappeared completely when treated with relatively low daily doses. In case 5 an extensive infection of the larynx was present, a condition that is supposed to lower the radiosensitivity. In case 13 no signs of infiltration of the cartilage or of the deeper layers of the musculature by tumor cells could be found and the fixation of the affected half of the larynx was apparently due to collateral edma. Histologically the tumor must be regarded as relatively radiosensitive, as was proved by its quick regression with a relatively low total dosage, 5,450 roentgens. In stating that the lesions in these 4 cases were anatomically healed we do not necessarily mean a definite anatomic and clinical healing; we mean only that at the time of examination there was evidence neither of intact cancer cells in the larynx nor of metastases outside the larynx in the representative sections examined.

Two tumors (cases 4 and 6) were similar in histologic structure and site of origin to the 4 just mentioned. The tumor in case 4, however, extended into the musculature, and that in case 6, into the anterior part of the thyroid cartilage. The tumor in case 4 disappeared clinically after treatment but histologically showed morphologically normal-appearing cancer cells, while that in case 6 showed a less complete regression after irradiation. Conjecture as to why these 2 tumors did not respond equally well to radiation necessitates the conclusion that though there was no histologic difference and no special radioresistance of the tumor cells within the bone (for cells examined in this region were necrotic), the deeper infiltration of the tumor cells made them definitely less radiosensitive. Considerations of age, nutrition and other factors do not seem to enter the picture, inasmuch as these cases were similar in such respects to 2 of the cases in which cure was effected.

No definite relation between total dose, method of fractioning, radiosensitivity of the tumor and appearance of early fibrosclerotic transformation of the tissue leading to incurability of the tumor was found.

In 2 cases in which the roentgen therapy had to be prematurely interrupted, a rather marked regression of the tumor with histologic evidence of necrosis of the peripheral parts, especially that portion infiltrating the musculature, was present, and a question must arise as to what might have happened if roentgen therapy had continued.

In 3 tumors there was partial regression: one was a semiepidermoid carcinoma of the piriform sinus with metastases; another, an epidermoid carcinoma of the mucosa type in the vallecula; and the third, an epidermoid carcinoma of skin type in the ventricular fold, with extensive infiltration of the whole half of the larynx, involving the arytenoid cartilage and leading to complete fixation of the cord.

The undifferentiated tumors of the piriform sinus (cases 2, 9 and 11) reacted fairly well to radiation, but the tumors could not be completely destroyed. In 2 cases (2 and 9) the doses were apparently too small, but in the third, in which the total dose of radiation was sufficient, only clinical regression was accomplished and there was local recurrence after six weeks. The main cause for failure of radiation therapy in the third case, and in cases of tumor of the piriform sinus generally, may be the fact that the disease was in an advanced stage and that glandular metastases develop much more quickly in such cases than in cases of intralaryngeal tumors. In all 3 cases metastases were present before the onset of radiation.

COMMENT

In this series it was observed that protracted fractional irradiation causes in tumors in the early stages rather intensive necrosis of the surface epithelium, damage of the glandular epithelium, and inflammatory reaction mainly of the superficial layers of the mucosa. The latter reaction is characterized by intensive fibrinous exudation with formation of a pseudomembrane and vascular changes of a specific character. Later there is regeneration of the epithelium as well as more marked fibrosis of the superficial layers of the mucosa, with definite decrease in the subepithelial lymphatic infiltration.

There may also be some obliteration of the small and medium-sized vessels, scattered atrophy of the glandular lobules and frequently chronic edema. Occasionally there may be secondary involvement of the musculature by the fibrosis. However, there are no changes of the musculature, bone or cartilage primarily due to radiation. The reaction of the tumor tissue is inconsistent with the histologic structure but seems connected more definitely with the location of the tumor. The criterion of the location, however, is not sufficient to determine the exact results of irradiation of the tumor tissue.

SUMMARY

Sixteen cases of intralaryngeal and extralaryngeal neoplasms occurring in men from 48 to 78 years of age have been reviewed.

The reaction of the normal laryngeal tissues and of the cancer growth have been outlined.

Attention has been drawn to some of the unknown factors in roentgen therapy of tumors of the larynx.

Case Reports

SUBLINGUAL EPIDERMOID CYSTS

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Sublingual epidermoid cysts are uncommon. Shore[1] stated that only 4 have been observed in 54,000 surgical specimens examined in the pathologic laboratory of St. Luke's Hospital, New York, during the past forty years. The case here reported is of interest not only because of the infrequency with which the condition is encountered but also because during a period of thirty years a correct diagnosis was not made.

REPORT OF A CASE

J. D., a white man aged 48, a farmer, was admitted to the United States Veterans Administration Hospital on Oct. 12, 1938, with a diagnosis of "hypertrophy of the left sublingual gland." The history revealed that in 1936 two cervical vertebrae were fractured as the result of an injury.

At the age of 18 (thirty years ago) the patient became aware of a small sublingual mass on the left side. It did not give rise to symptoms but appeared to increase in size gradually. By 1926 the mass had enlarged to such an extent that there was difficulty in swallowing. His family physician made a sublingual incision and evacuated a large quantity of foul yellowish sticky material. Within a year the mass had again become enlarged to its original size and was now presenting externally in the left submaxillary region. From 1926 to 1938 the patient was subjected to six external surgical operations, each of which evidently consisted of simple incision and drainage. After each operation the mass recurred.

General physical examination and laboratory studies of the blood, urine and sputum indicated no significant deviations from the normal. On examination of the mouth a mass was noted beneath the left half of the tongue. It was approximately the size of a large walnut. On bimanual examination it felt rather "doughy." The mucous membrane covering the mass was normal in appearance. No salivary calculi could be detected by palpation.

Externally the neck presented two scars, the residuals of previous operations. One small cicatrix, about 1 cm. long, was noted in the midline above the hyoid bone, while another scar, 4 cm. long, was found parallel to the ramus of the mandible, over the left submaxillary gland.

Roentgen ray studies failed to show the presence of calculi or other opaque bodies.

A fine probe was easily passed into the submaxillary duct without encountering any obstruction. It was then decided to incise the mucous membrane over the mass in order to determine its nature and extent. Anesthesia was obtained by placing a small pledget of cotton, moistened with 2 per cent pontocaine hydrochloride solution, over the mass under the tongue. The overlying mucous membrane, in addition, was infiltrated with a 2 per cent solution of procaine hydrochloride

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The United States Veterans Administration does not assume responsibility for statements made or opinions expressed herein.

1. Shore, B. R.: Sublingual Epidermoid Cysts, Ann. Surg. **108**:305 (Aug.) 1938.

containing 8 minims (0.5 cc.) of epinephrine to the ounce (30 cc.). A transverse incision was made, and the dome of the mass was exposed by undermining the mucous membrane with scissors. The wall of the sac was yellowish and firm. It did not have the cystic feel of a ranula. A knife was inserted, and immediately a large quantity of foul yellowish cheesy material spurted out in a steady stream, much after the manner of tooth paste coming out of a tube on pressure. The incision was enlarged, and several ounces of the contained secretion was scooped out. By insertion of the index finger, the limits of the sac were determined. It was obvious after a brief examination that it was too large for safe intraoral removal, in view of the fact that secondary infections and reactions from previous surgical explorations had obliterated the cleavage lines. Anteriorly it extended almost to

Fig. 1.—Lateral roentgenogram showing an epidermoid cyst filled with iodized poppyseed oil after evacuation of contents.

the symphysis mentis, while posteriorly it reached to the hyoid bone. Inferiorly and anteriorly the wall of the sac was adherent to the skin of the left submaxillary region, where it had been incised previously. A stab wound was made into the sac through the old healed external scar, and the oral incision was closed.

Two days later the sac was irrigated with sterile physiologic solution of sodium chloride through the external wound, and 20 cc. of iodized poppyseed oil 40 per cent was injected into it. The opening was temporarily plugged with a pledget of cotton. Lateral and anterior roentgenograms confirmed the anatomic limits determined previously by digital exploration.

Three days later, under anesthesia obtained by cervical paravertebral block and local infiltration with 0.5 per cent procaine hydrochloride solution, the sac was approached externally through an incision 5 cm. long in the left submaxillary region, parallel to the ramus of the mandible. The skin and the platysma muscle were incised. The anterior belly of the digastric muscle was retracted and the mylohyoid muscle incised at a right angle to its fibers. The sac presented,

but because of strong adhesions which welded it to the surrounding structures, it was not possible to find a line of cleavage. It was therefore removed in sections; the method utilized was chiefly sharp dissection. The wound was closed in layers, and a strip of rubber tissue was inserted for drainage.

Healing took place without incident. Four months later the patient reported no evidence of recurrence.

COMMENT

Sublingual epidermoid cysts result from the inclusion of ectoderm in the mesenchymal area during the developmental period.[2] This occurs, as a rule, at the point of junction of the first three branchial clefts in

Fig. 2.—Photomicrograph under low power showing the histologic structure of an epidermoid cyst: *A*, epidermal lining; *B*, keratohyaline material in cyst; *C*, dense supporting fibrous tissue. Note absence of papillae and accessory skin structures.

the midline, which corresponds to the submental region in the adult. In this position the epidermoids may later become manifest either beneath the skin or between the geniohyoid muscles. Occasionally an epidermoid cyst may appear laterally below the angle of the jaw. In this situation it is to be distinguished from a branchial cyst. More frequently an epidermoid cyst arising in the midline may, by enlargement, encroach laterally.

2. Semken, G. H.: Surgery of the Neck: Sublingual and Submaxillary Cysts, in Nelson New Loose-Leaf Surgery, New York, Thomas Nelson & Sons, 1937, vol. 2, p. 802.

The wall of the epidermoid cyst is composed of a stratified epithelial lining supported by a layer of dense fibrous tissue.[3] The rancid cheesy material usually found in the cyst represents the accumulated secretion of sebaceous glands. In some cases hair follicles and sweat glands have been found, but other structures of ectodermal origin have not been found in the cases reported.

DIAGNOSIS [4]

The sublingual epidermoid cyst is to be distinguished from the ranula, which results from cystic degeneration of the sublingual gland. The ranula is reddish or bluish gray and has a distinct fluctuant, elastic feel on palpation. The sublingual dermoid cyst, because of its thicker wall, is distinctly doughy on pressure and has a yellowish tinge. Ranulas, in contrast to dermoid cysts, are thin walled and lined with cuboidal epithelium. They are adherent to adjacent structures and contain a fluid resembling saliva. They are, as a rule, difficult to remove without rupture.

Secondary infection of a dermoid cyst, not infrequently resulting from simple intraoral drainage, may produce a confusing picture resembling that of Ludwig's angina or that of a fungating malignant growth.

Sublingual thyroid tissue may require differentiation from dermoid cyst. Hypertrophy of the submaxillary glands, adenomas and neoplasms, both benign and malignant, involving the structures of the floor of the mouth, must be considered. Thyroglossal and branchial cysts should offer no difficulty.

A sublingual dermoid cyst does not become manifest until early adult life. At that time a smooth, circumscribed, nonadherent painless mass may be noted in the floor of the mouth. It may eventually attain the size of an egg and then shift laterally. There may be a visible external bulge in the submental region, or it may push the tongue upward. A distinct yellowish tinge may be noted through the mucosa.

TREATMENT [4]

Treatment consists of complete excision. Where the cyst presents in the floor of the mouth, intraoral removal is not difficult, because, if secondary infection has not occurred, a distinct line of cleavage can be utilized for blunt dissection. The thick fibrous wall of the cyst permits traction without danger of rupture. If repeated or extensive secondary infection has occurred, as in the case reported, external incision with adequate exposure is necessary. Adherence in certain areas may make sharp dissection necessary. As Shore [1] pointed out, the injection of irritating or sclerosing solutions into an area of mesenchymal origin for purposes of obliteration can be understood. In a cavity lined with epithelium, such as a dermoid cyst, every vestige of epithelium would have to be destroyed to assure the success of such a method. Surgical excision is therefore the treatment of choice.

United States Veterans Administration.

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3. Padgett, C. C.: Diseases of the Mouth and Jaws, Philadelphia, W. B. Saunders Company, 1938, pp. 484-488.

4. Blair, V. P.: Surgery and Diseases of the Mouth and Jaws, St. Louis, C. V. Mosby Company, 1912, pp. 484-490.

GLOSSOPHARYNGEAL NEURALGIA

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The infrequency of instances of glossopharyngeal neuralgia prompts the reporting of this case. Previous excellent reviews of the subject obviate a lengthy discussion in this paper.[1] The syndrome is similar to that of trigeminal neuralgia, except for the distribution of pain. There are sudden paroxysms of pain, originating in the side of the throat or deep in the ear or in the base of the tongue. The pain radiates almost always to some part of the ear. Swallowing and the raising of the pressure in the intrapharyngeal region or the intraeustachian tube (as in sneezing) are common precipitating acts. The paroxysms tend to recur, and at shorter intervals. There may or may not be a demonstrable trigger zone. There is no known cause or pathologic condition associated with this syndrome, though of course ninth nerve pain may result from a traumatic or a neoplastic lesion.

REPORT OF A CASE

The patient (C. A. R.) was a man 44 years of age. His chief complaint was of pain deep in his left ear, this pain having first occurred six months previously, immediately after he drank a bottle of a cold soft beverage. He had had no further pain until one month before he was seen by us and during the past month had had numerous mild attacks, with severe attacks intermingled. The pain seemed to start in the side of his throat, at about the junction of his throat and ear on the left side, and then shot deep into his ear. It was usually initiated with the beginning of swallowing. Attacks, however, had been brought on by the turning of his head and occasionally occurred when he talked. At times the pain was extremely severe and was likened to that from a knife jab. He recalled that it had been brought on by blowing his nose.

Numerous physicians had made various diagnoses, and several types of therapy had been advised and carried out, including extraction of a tooth, ingestion of large amounts of vitamin B and chiropractic treatments. The severe pain persisted, however.

General physical examination of the patient revealed no abnormal conditions of note. The results of neurologic examination were essentially negative.

In view of the tentative diagnosis of glossopharyngeal neuralgia, based on the type of pain (attacks of sharp, shooting character), its location (in the distribution of the ninth cranial nerve) and the feeling that this was not a case of trigeminal neuralgia, the patient was examined by one of us (C. K. S.) with the intention of determining, if possible, a trigger zone and of attempting to stop the pain by anesthetizing such a trigger point.

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1. Dandy, W. E.: Glossopharyngeal Neuralgia, Arch. Surg. **15**:198-214 (Aug.) 1927. Peet, M. M.: Glossopharyngeal Neuralgia, Ann. Surg. **101**: 256-268 (Jan.) 1935.

In searching for a trigger zone it was necessary to attempt to anesthetize the various areas supplied by the ninth nerve. Briefly, the first branch of importance is the tympanic nerve, which forms, along with branches of the carotid plexus of the sympathetic nerve on the internal carotid artery, the tympanic plexus for the supply of the mucous lining of the tympanum, mastoid cells and auditory tube. In the neck there is a branch, the stylopharyngeus, which supplies the stylopharyngeus muscles, the only motor branch of this nerve. The pharyngeal branches of the nerve supply the mucous membrane of the dorsal third and the lateral half of the tongue and the front of the epiglottis, and a tonsillar branch supplies the mucous membrane covering the palatine tonsil and adjacent parts of the soft palate and the palatine arches.

Eucupin (isoamylhydrocupreine) 2 per cent was injected directly through the drum membrane into the middle ear. At this time the patient was having spasms of pain, which came regularly every three minutes. No relief was afforded by the procedure described. A 2 per cent solution of pontocaine hydrochloride was introduced into the eustachian canal through a eustachian catheter. The attacks persisted uninterruptedly. Next, the areas on the tongue previously described were cocainized, with no effect. Finally, as the applicator used in cocainizing the mucous membrane of the pharynx touched a point on the upper part of the pharynx directly posterior to the eustachian orifice and high up on the lateral pharyngeal wall, an attack was immediately brought on, and this area was extremely sensitive. Repeated attempts to cocainize the area were met with a violent reaction of pain and immediately precipitated a new attack.

It should be mentioned that cocainization of the sphenopalatine ganglion had no effect of any kind, either in initiating the attack or in lessening the pain.

We felt that the definite establishment of a trigger zone, as described, even though anesthetization of the area did not stop the attacks of pain, clinched the diagnosis of glossopharyngeal neuralgia.

The patient was admitted to the University of Kansas Hospitals on April 9, 1940, and on April 11 was operated on. With the patient under anesthesia produced by avertin with amylene hydrate and ether (intratracheal), a unilateral cerebellar approach was made on the left side. The bone was removed by means of a rongeur laterally to the edge of the mastoid region and mesially to permit exposure of the lateral recess of the cisterna magna. The dura was incised and the cerebellum retracted to permit an opening to be made into the cisterna magna, and a large amount of fluid was removed by suction. The cerebellum was then retracted posteriorly and the left lateral part of the medullary cistern exposed and opened. This procedure permitted the escape and the removal of a large amount of cerebrospinal fluid. The ninth, tenth and eleventh cranial nerves now were easily visualized, proximal to their exit through the foramen of the jugular vein. No abnormal conditions were visible, and the ninth nerve was lifted away from the other two, crushed and divided. Closure of the wound was carried out in layers, with interrupted black silk sutures.

The patient had an uneventful postoperative course and was up and out of bed on the tenth postoperative day. He had no pain in his throat or ear after the operation. He did mention a "drylike" taste in his mouth, which may well be a result of interruption of ninth nerve fibers to the parotid gland with an alteration in its secretion.[2]

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2. Erickson, T. C.: Paroxysmal Neuralgia of the Tympanic Branch of the Glossopharyngeal Nerve, Arch. Neurol. & Psychiat. **35**: 1070-7075 (May) 1936.

At present the patient describes a bitter taste in the mouth on the affected side. He is now required to put much more sugar in his coffee or iced tea to get the taste he previously had. The bitter taste may change suddenly into a sweet taste. He feels that there is a definte lessening of the amount of saliva in the mouth. Of course, the areas on the tongue and pharynx which are supplied by the ninth nerve are completely numb. Hearing, swallowing and other functions are not affected.

Clinical Notes; New Instruments and Technics

SIMPLIFIED METHOD OF REMOVING A TRIANGULAR WEDGE OF NASAL BONE IN THE PLASTIC CORRECTION OF LATERAL DEVIATION OF THE NOSE

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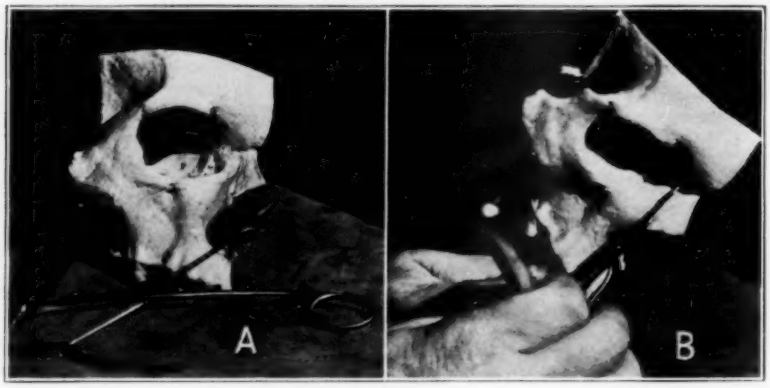
In the plastic correction of lateral deviation of the nasal axis, which is almost invariably a traumatic condition, it is required to move the nose back to the midline. When the deviation is slight, section through the nasofrontal process by the usual intranasal incision will usually permit replacement to the midline, involving only a slight overriding of the frontal process of the superior maxillary bone.



Photographs showing (*A*) the clamp and saw and (*B*) the method of applying the instruments.

In cases of marked deflection, it becomes necessary to remove a triangular wedge of bone from the broad concave side in order to create space for shifting the narrow convex side medially without marked overriding of bone.

TECHNIC

After preliminary antisepsis and the production of anesthesia, a curved double-edged knife is entered at the lateral angle of the apertura piriformis (on the broad concave side of the nose) and directed upward over the outer surface of the nasal bone toward the inner canthus. Care is taken to keep the tip of the knife close to the bone, to avoid trauma to the soft tissues and to minimize postoperative discoloration. The knife is removed but is reintroduced through the same incision in the same direction and is directed along the under side of the same bone. A periosteal elevator is then inserted, elevating the membrane from both sides of the bone. An angulate saw is next inserted. The cutting edge is placed along a line from the outer angle of the apertura piriformis to a point midway between the inner canthus and the root of the nose, and section is made. A wedge-shaped

defect is then created by reinserting the saw in a more anterior position—the deep end at the upper extremity of the first line of section but the proximal end anterior to the lower end of this line—creating a triangle with its base on the unattached border of the nasal bone and its apex at the root. The width of the base is naturally proportionate to the amount of deviation.

In making this second section through the nasofrontal process, one usually encounters difficulty in keeping the saw in an imaginary line at a fixed angle to a line of section that is invisible. Slight angulation will distort the relation of the two lines, creating difficulty in the removal of a complete one piece wedge.

I have overcome this difficulty with a simple technic, to wit, the use of a straight clamp, the approximating surface of which is triangular, and a saw, the handle of which is at right angles to the cutting edge. After the first section through the nasofrontal process is made, as previously described, and before the saw is removed, the clamp is inserted and fixed on the plate of bone flush with the anterior surface of the saw. The saw is then removed and reinserted with the cutting edge flush with the anterior border of the clamp, and section is made with the clamp as a guide. When section is completed, the clamp has a good hold on the entire wedge, precluding the danger of breaking off parts of the wedge. By using clamps of varying widths, it is possible to select one which closely approximates the width of the wedge to be removed.

The accompanying photographs show the instruments used and the method of application.

Abstracts from Current Literature

Ear

SUMMARY OF ROUND WINDOW GRAFT OPERATIONS PERFORMED FOR DEAFNESS. WALTER HUGHSON, Ann. Otol., Rhin. & Laryng. **49**:384 (June) 1940.

The author reviews the results of experiments, using the Wever and Bray phenomenon, on the function of the round window in auditory acuity and discusses the theories regarding its function. He feels that the most plausible theory is that at least one function is to dampen the intensity of acoustic stimuli reaching the perceptive mechanism of the internal ear. He feels that permanent surgical fixation of the round window would eliminate this dampening and, to that degree, improve hearing on the side of the operation. The operation was performed first on cats, and grafts of fascia, muscle and periosteum were used. The author has since operated on 35 human subjects in somewhat more than three years, with no complications developing and with enough improvement gained to warrant further work along the same lines. The patients must be carefully selected and should not be above 50 years of age, with a loss of hearing not greater than 50 decibels in the speech frequency range. He feels that a moderate degree of nerve deafness, as demonstrated by bone conduction and loudness balance tests, need not prove a contraindication.

M. V. MILLER, Philadelphia.

RESULTS OF THE LABYRINTHINE FISTULIZATION OPERATION FOR CHRONIC PROGRESSIVE DEAFNESS: REPORT OF CASES. EDWARD H. CAMPBELL, Ann. Otol., Rhin. & Laryng. **49**:447 (June) 1940.

In order to obtain favorable results it is necessary to give attention to three factors: proper selection of the patient, proper operative technic and proper postoperative care. In regard to the first, the author says that the initial deafness should not be greater than 60 to 65 per cent, but he feels that if other factors remain favorable a deafness of 70 to 80 per cent is not a contraindication. However, an improvement of 30 to 35 decibels at the critical levels still will not bring the hearing to a practical level, although such improvement may help the patient, particularly if the tinnitus is lessened. Bone conduction should be good if good results are to be expected. Normally functioning vestibular mechanism is felt to be necessary. A healthy condition of the cutaneous surface of the external wall of the canal, particularly that of the upper and posterior areas, seems essential. Exact knowledge of the anatomy and painstaking adherence to proper technic are prime requisites. Negligence in postoperative care may cause failure in an otherwise successful treatment. Important to the success of the treatment are maintenance of asepsis and proper control of granulations with a 25 per cent solution of trichloroacetic acid or silver nitrate. Good general care is necessary and the avoidance of too much stimulation of the labyrinth by exertion. Eight cases are reported and analyzed. In 3 the condition showed over 20 per cent improvement eight to fourteen months after operation. The results in the others were not satisfactory; 2 of the patients, it is realized now, were unsuitable for operation, both having decreased bone conduction and 1 having a dead labyrinth. Two fistulas closed promptly after operation. The results obtained, however, serve to emphasize the importance of the three factors mentioned first.

M. V. MILLER, Philadelphia.

THE USE OF PRESERVED CARTILAGE IN EAR RECONSTRUCTION. HAROLD L. D. KIRKHAM, Ann. Surg. **111**:896 (May) 1940.

Experiments on rabbits demonstrated that heterologous grafts remain as cartilage, the cartilage retaining its form even though cellular death has occurred.

Transplantation of aural cartilages from cadavers which had been stored in the solution recommended by O'Connor and Pierce, consisting of aqueous solution of merthiolate in physiologic solution of sodium chloride, 1:4, has been successfully accomplished, with loss of the graft in only 1 case. The reconstruction of the ear is accomplished in about five stages. At the first stage the cartilage, which has been previously perforated, is planted under the skin of the mastoid area through a curved incision high in the hair line. In about two months the cartilage, with the skin of the mastoid region, is raised, thus bringing the new ear forward, leaving a raw surface over the mastoid and back of the new ear. The area is covered with a thin split graft placed on a molded stent. At a later date a double pedicle tube flap is made on the neck, about the size of a lead pencil and long enough to be carried over the ear. In about three weeks the lower end of the flap is transferred to the edge of the new ear, and in three more weeks the draping of the flap around the new ear to form the helix is completed and the excess removed or returned to its bed. It is believed that ears so constructed, with a normal ear scaffold, are more sightly and more satisfactory to the patient and the surgeon alike.

H. Miller, Philadelphia.

The Labyrinth and Experimental Beriberi. G. Vercellana, Arch. ital. di otol. **52**:61 (Feb.) 1940.

It is known that polyneuritis in birds is often accompanied by phenomena resembling disturbances of equilibrium from lesions of the labryinth, and many investigators have made histologic studies of the labyrinths of pigeons with avitaminosis B_1. Vercellana made comparative clinical studies of 126 pigeons, some with unilateral and some with bilateral labyrinthectomy, which, with adequate controls, were placed on a diet of polished rice for a sufficient length of time for beriberi to develop. The birds were examined with reference to the presence or absence of the six fixed positions of Ewald. Some were blindfolded. Copious details and numerous photographs of the birds experimented on are presented. The conclusions are that bilateral ablation of the posterior labyrinth of pigeons does not modify the phenomena of beriberi, produced by the exclusive administration of polished rice, even when the optic reflex of position is abolished (to eliminate the doubt of an eventual vicarious labyrinthine function). These observations support the assumption that there is no direct relation between the labyrinth and experimental polyneuritis.

Dennis, San Diego, Calif.

Disposition of the Eustachian Tube in Guinea Pigs. C. Calabresi, Arch. ital. di otol. **52**:89 (Feb.) 1940.

Calabresi noted that in some cases serial sections of the tympanum of the guinea pig show the tubal cartilage thrust deeply into the median wall of the tympanic cavity. No reference to this was found in the literature. In this article Calabresi describes the placement and structure of the tube in guinea pigs. The tube terminates in a blind end, situated on the internal face of the lateral wall of the tympanic cavity near the basal turns of the cochlea.

Dennis, San Diego, Calif.

Complementary Functions of the Osseous and Aerotympanic Pathway in the Transmission of the Sound Wave for Its Reception and Extinction. P. Brisotto, Boll. d. mal. d. orecchio, d. gola, d. naso **58**:3 (Jan.); 41 (Feb.) 1940.

Brisotto claims that physical and psychophysical phenomena are combined to regulate the reception and perception of sound and that the latter are developed normally only if, the sensation having been received and recorded, the auditory apparatus is brought back into balance by the active extinction of superfluous tympanic and labyrinthine tones. On the basis of these suppositions, he divides the acoustic phenomenon into three phases: transmission, sensation, or perception, and extinction.

He states that a sound wave causes the tympanic membrane to vibrate and passes along the ossicular chain to bring the acoustic impulse to the perilymph and endolymph, which pass it on to the neuroepithelium of the cells of Corti with a velocity about three times that of that portion of the same sound wave which travels across the air through the tympanic cavity and beats against the membrane of the fenestra rotunda. The acoustic impulse which has traveled the ossicular chain, having been received by the labyrinthine fluids, sets up a current in the labyrinth, a portion of which he claims is spent in bringing about a pause by counteracting on the labyrinthine side of the fenestra rotunda the effects of the wave arriving by the aerial route at the membrane. He states that the latter wave must be considered superfluous and might even be damaging in its effects, because it might create interference or even produce two sensations were it not elided by a mechanism which is immediately set into motion when the wave, traveling by way of ossicles, is perceived. This mechanism is in part physical (the action of the superfluous intralabyrinthine wave reaching the membrane of the fenestra rotunda) and in part a reflex action, both actions employed to bring about an extinction of the sound wave.

Brisotto states that the position of the footplate of the stapes varies in direct relation to the intensity and direction of the sound wave. The sound wave carried by air undergoes variations of phase, such as the loss of a half period, by the reboundings from the walls of the tympanic cavity and because of the distance traveled, to the extent that when it finally strikes the membrane of the fenestra rotunda it is of a directly opposite phase to the intralabyrinthine wave. By this reasoning, two residual waves meet each other with equal intensity but in opposite phases and so automatically elide each other. In such a fashion, these two residual waves contribute to the extinction of the whole wave; thus, a pause ensues, allowing a more perfect reception of the succeeding wave by the auditory apparatus.

He further states that if pathologic changes, such as destruction of the tympanic membrane or the ossicular chain and changes in the mucous membrane of the middle ear (particularly if located in the region of the two windows), are present, the loss of hearing is due in great part to the loss of ability of the auditory apparatus to extinguish the sound wave before a succeeding sound wave arrives, thus permitting an overlapping of the succeeding sound wave and producing confusion thereby.

In summarizing, the author bases his reasoning on laws of physical acoustics and states that the phenomenon of hearing relative to the transmission of sound waves to the labyrinth cannot be only by bone or only by air. Necessarily, if by physical laws both must transmit tones and the aerial transmission is retarded, this must occur for functional reasons.

Studying the latter phenomenon, the author develops a theory of the complementary ways that the aerial roadway transmits the retarded wave for the extinction which is necessary between tones and which is synchronized with the same tone that has gone by the osseolabyrinthine way. He says that his arguments are supported by embryologic study and comparative anatomy.

He illustrates the play of the contractions of the tensor tympani due to the action of its double innervation and shows that it has two distinct functions—one related to the sinking of the stapes (in direct proportion to the intensity of the tone) and one which coincides every one-fifteenth second with the extinguishing process.

He states that this theory illustrates the admirable harmony of the functions combined with mathematical precision, and he seems to find in recent papers on the electrophysiology of the ear confirming evidence of the complementary action of the two way transmission of a sound wave—aerial and osseous.

In this fashion, the theories of Cotugne and Scarpa would be fused into one single theory and their combined actions explained on the basis of physical and psychophysical laws, which, he claims, necessarily dominate every functional phenomenon, particularly in an organ so highly differentiated as the ear.

MORICONI, Trenton, N. J.

ALLERGIC IRRITATION OF THE INTERNAL EAR. CHARLOTTE MEYER, Pract. oto-rhino-laryng. **3**:92 (April) 1940.

The author discusses the possibility of allergic reactions affecting the labyrinth and the internal ear, particularly from the standpoint of disturbances of the vago-sympathetic system. Whether or not this reaction assumes a form of angioneurotic edema or of another type of neurovascular disturbance is immaterial. However, she states that many symptoms resembling those of Ménière's disease are definitely traced to an allergic base.

She cites a number of cases of vestibular irritation occurring in persons who were definitely allergic, whether with bronchial asthma or with vasomotor rhinitis, and in whom labyrinthine symptoms, including vertigo, tinnitus, occasional deafness, nausea and vomiting, occurred. These symptoms were usually transitory and responded to routine allergic therapy.

In summarizing, the author states the belief that there exists a true allergy of the internal ear, manifested as an edema which can act on various parts of the labyrinth or the cochlea. Hence, in cases of Ménière's disease, particularly in those in which the genesis is not completely clear, careful attention and thought should be given to the possibility of an allergic background. She also stresses the need for a thorough taking of the history and physical examination and emphasizes the point that the skin tests are not always dependable and do not indicate the kind or degree of hypersensitivity which is present.

PERSKY, Philadelphia.

Larynx

THE MANAGEMENT OF ORGANIC FOREIGN BODIES IN THE TRACHEA AND BRONCHI OF CHILDREN—ILLUSTRATED WITH LANTERN SLIDES. ELBYRNE G. GILL, Virginia M. Monthly **66**:275 (May) 1939.

Thirty-six cases are reported in which a vegetable foreign body was aspirated into the trachea or a bronchus. The patients were aged 7 months to 11 years. The foreign bodies in the order of frequency were peanuts, walnuts, corn, beans, chestnuts, apple, coconut and watermelon seed. All were removed bronchoscopically except two which were removed through tracheotomy wounds and four which were removed by coughing. Emphasis is placed on the importance of obtaining a careful history of cases of unexplained cough and irregular but persistent fever in children. Roentgen examination and careful physical examination should be made, and in the presence of signs and symptoms suggestive of a foreign body, bronchoscopic procedures should be used. The three signs most often found are (1) unilateral limitation of expansion of the lung and chest on the affected side; (2) hyperresonance on percussion, and (3) diminution or total absence of the breath sounds.

For anesthesia during bronchoscopic procedures the author uses sodium amytal administered by bowel, together with ether given with extreme caution by the drop method.

As to complications, tracheobronchitis present prior to operation continued in 6 cases and bronchopneumonia occurred in 2 cases. Tracheotomy was done in 9 cases—to relieve postoperative edema, dyspnea prior to or immediately following bronchoscopic procedures and in 1 case for drainage of a pulmonary abscess. In the 36 cases there were 35 recoveries. The 1 death was due to general sepsis secondary to pulmonary abscess in a child admitted to the hospital in a moribund condition.

STONE, Richmond, Va. [AM. J. DIS. CHILD.]

TWO CASES OF FATAL CONGENITAL STRIDOR IN A FAMILY. M. NAGEOTTE-WILBOUCHEWITCH, Bull. Soc. pédiat. de Paris **37**:85 (Feb.) 1939.

Two members of a family of 8 children died in infancy of bronchopneumonia. Both had laryngeal stridor, brassy cough and occasional vomiting. Roentgeno-

grams revealed nothing in the chest to account for the stridor, and bronchoscopic examinations showed no abnormality in or above the larynx. It is thought some tracheobronchoesophageal anomaly was present. No autopsies were performed.

BENJAMIN, Montreal, Canada. [AM. J. DIS. CHILD.]

ASPIRATION OF A TOY IN CHILDHOOD. H. LÖFFLER, München. med. Wchnschr. **86**:260 (Feb. 17) 1939.

Löffler reports the case of a 13 month old infant who aspirated a toy nipple, 2 cm. in length. The infant cried out and collapsed and was immediately rushed to the hospital but was dead on arrival. Autopsy revealed the nipple obstructing the larynx. The nipple was the kind attached to toy nursing bottles for dolls. Löffler reemphasizes the danger of permitting little children who put things in their mouths to play with small toys.

BRAHDY, Mount Vernon, N. Y. [AM. J. DIS. CHILD.]

Nose

MECHANISM OF ESTROGEN EFFECT ON NASAL MUCOSA IN ATROPHIC RHINITIS; SUCCESSFUL TREATMENT WITH PROSTIGMIN. S. SOSKIN and L. B. BERNHEIMER, Proc. Soc. Exper. Biol. & Med. **42**:223 (Oct.) 1939.

Fourteen patients suffering from advanced atrophic rhinitis with ozena were treated from two to six months by spraying 0.25 cc. of a 1:2,000 solution of prostigmine methylsulfate into each nostril four times daily. A nasal irrigation with a mild alkaline fluid was given once or twice daily. All patients definitely improved within two weeks; crust formation decreased, as did the nasal discharge, and the ozena was controlled. The results were far superior to those from local treatment with estrogenic substance. Previous studies disclosed that the benefit from estrogenic substance in the nasal mucosa was due to (1) specific proliferative effects and (2) effects secondary to hyperemia. The acetylcholine-liberating property of estrone (theelin) probably accounted for the benefit from intranasal estrone therapy in atrophic rhinitis, which thereby produced hyperemia. The authors then reasoned that acetylcholine effects could be obtained by inhibiting the choline esterase, thus the use of a synthetic physostigmine-like substance in this study.

HANSEN, Minneapolis. [AM. J. DIS. CHILD.]

PHYSIOTHERAPY IN THE COMMON COLD AND SINUSITIS. W. K. RUSSELL, Brit. J. Phys. Med. **2**:92 (April) 1939.

Russell stresses the importance of ultraviolet irradiaton in the prevention of colds and the local use of ultrashort wave therapy in the treatment of colds and sinusitis. His statistical evidence was obtained from other workers.

HARTMAN, Cleveland. [AM. J. DIS. CHILD.]

MAXILLARY SINUSITIS IN THE SMALL CHILD. J. C. OREGGIA, Arch. de pediat. d. Uruguay **10**:197 (April) 1939.

Two forms of maxillary sinusitis are described, an osteomyelitic form in the young child and a purulent form in the older child, usually following infectious diseases.

Oreggia observed 3 patients with the osteomyelitic form, all children 6 years of age. The condition in each child was due to staphylococcic infection. Two of the children responded to surgical treatment; the third child died, the condition being of a malignant type. The classic symptoms of the osteomyelitic form are: marked swelling of the internal angle of the eye and of the upper jaw accompanied by profuse nasal drainage.

The author has seen no definite connection between adenoiditis and sinusitis. Many of the children with sinusitis are children who are affected with a constitutional diathesis. These children continue to have sinus disease in spite of the removal of tonsils and adenoid vegetations. They suffer from chronic and mucopurulent catarrh. Often they have prolonged subacute febrile manifestations. There is usually much postnasal drainage. Often the mucous membranes of the nasal fossae are pale and atrophic.

Removal of the adenoids does not always cure the sinusitis and the prevailing repetitional attacks.

The author advises against puncture of the maxilla and washing of the sinuses. If a permanent window has to be made, the gingivolabial route is preferred to the intranasal. As a local treatment the author advises nasal shrinkage with cocaine and suction, together with local use of antiseptics or antivirus preparations.

SCHLUTZ, Chicago. [AM. J. DIS. CHILD.]

Miscellaneous

SULFANILAMIDE AND MENINGITIS. S. W. SAPPINGTON and GRANT O. FAVORITE, Ann. Int. Med. **13**:576 (Oct.) 1939.

The cases of 227 patients with meningitis are reviewed. Those in which the hemolytic streptococcus and the meningococcus were the causative organisms showed the best response to sulfanilamide therapy. The results in cases of pneumococcic meningitis were better than with previous types of treatment employed. Sulfanilamide had little or no effect in influencing the course of influenzal and tuberculous meningitis. READING, Galveston, Texas. [AM. J. DIS. CHILD.]

ORAL RAGWEED POLLEN THERAPY. M. ZELLER, J. Allergy **10**:579 (Sept.) 1939.

Zeller concludes from his experiments that enteral absorption of ragweed pollen was not demonstrated by passive transfer and direct skin tests. He therefore sees no advantage in oral therapy and feels that the best results are obtained by the hypodermic method. However, among those who discussed his paper were some who still held hope for oral therapy. They contended that the fact that some persons manifest constitutional symptoms following ingestion of pollen extract and the fact that a proteose has been demonstrated in the urine of patients who ingested pollen extract cannot be disregarded. HOYER, Cincinnati. [AM. J. DIS. CHILD.]

EPINEPHRINE IN OIL: ITS EFFECT IN THE SYMPTOMATIC TREATMENT OF HAY FEVER. E. L. KEENEY, J. Allergy **10**:590 (Sept.) 1939.

Injections of 0.2 to 0.5 cc. of epinephrine in oil were given to 35 adults who had hay fever. Relief of symptoms varying from 25 to 100 per cent was obtained in 30 of the 35 patients. The effect lasted twelve to thirty-six hours. A few of the patients experienced nervousness for one to three hours. This experience was prevented subsequently by reducing the dose.

HOYER, Cincinnati. [AM. J. DIS. CHILD.]

NOTE ON ORAL POTASSIUM CHLORIDE THERAPY IN ASTHMA, HAY FEVER, URTICARIA, AND ECZEMA. DAVID HARLEY, J. Allergy **11**:38 (Nov.) 1939.

Potassium chloride, in doses of 5 grains (0.32 Gm.) dissolved in a glass of water and given orally one-half hour before meals three times a day, failed to produce a significant degree of improvement in a group of 43 allergic patients. These included patients with hay fever, allergic rhinitis and asthma, 10 with each, 9 patients with urticaria and 4 patients with eczema. The average duration of treatment was from two to three and four-fifth weeks.

One patient with urticaria, in whom there was moderate improvement, relapsed in four weeks but responded favorably to the administration of sodium chloride tablets three times a day in water.

MENDELSOHN, New York. [ARCH. DERMAT. & SYPH.]

CAUSE OF REDUCTION OF SUGAR CONTENT OF CEREBROSPINAL FLUID IN MENINGITIS. E. HENDRY, Arch. Dis. Childhood **14**:307 (Dec.) 1939.

Reduction in the dextrose content of the cerebrospinal fluid is a familiar observation in patients with meningitis of all forms, but the cause has not yet been definitely determined. From a review of the literature it seems unlikely that lessened permeability of the choroid plexus to the passage of sugar into the cerebrospinal fluid is the cause of the reduction. The evidence points, rather, to the action either of bacteria or of leukocytes. The author devised tests to determine if there is an alteration in the permeability of the blood-brain barrier to dextrose, if the action of bacteria is the cause of the reduction or if the presence of leukocytes is responsible for the reduction. He observed that in the early stages of cerebrospinal fever a greatly increased cell count and a positive bacterial culture are almost invariably encountered. At this time the sugar content is extremely low. The patients were treated with sulfanilamide, and sterile cerebrospinal fluid was found on the second, or at the latest on the third, occasion on which lumbar puncture was performed. As at this stage of the illness the cell count is still high and the sugar content, though rising, well below normal limits, these observations suggest that meningococci are not responsible for the breakdown of dextrose. In cases of meningococcic meningitis the cell picture of the fluid changes from one almost entirely of polymorphonuclear cells to one largely of lymphocytes. In the early stages there is great reduction in the sugar content of the cerebrospinal fluid, while in the later stages, when the exudate is largely composed of lymphocytes, the sugar content approaches the normal level. In conditions such as benign lymphocytic meningitis, in which there is a purely lymphocytic exudate, there is no reduction of sugar in the spinal fluid. In 2 cases of meningitis due to Bacillus coli there was complete absence of sugar in the cerebrospinal fluid, an occurrence which might be expected, as two factors are operative: a strongly glycolytic organism and a large polymorphonuclear cellular exudate. In the case of tuberculous meningitis conclusions are not so easily drawn, but it is suggested that, as the tubercle bacillus has practically no power to break down sugar and as it is present in the cerebrospinal fluid only in small numbers, the part that it plays in reducing cerebrospinal fluid sugar need not be considered. In the early stages of the disease the cellular exudate in the spinal fluid is largely lymphocytic, and at this time the sugar content of the fluid is not greatly below normal. As the disease progresses to its fatal issue there are an increase in the number of polymorphonuclear cells and a simultaneous decrease in the sugar content. It is concluded that polymorphonuclear leukocytes possess powers of glycolysis and that their presence accounts for the reduction of sugar in the cerebrospinal fluid in purulent meningitis and for the progressive decrease in tuberculous meningitis.

J. A. M. A.

RARE CAUSES OF COMPRESSION OF THE INTRACRANIAL PORTION OF THE OPTIC NERVE. G. P. SOURDILLE and M. DAVID, Ann. d'ocul. **176**:756 (Oct.) 1939.

Sourdille and David report the case of a patient with ocular symptoms characteristic of an optochiasmatic attack and auditory signs of an acoustic neurinoma. Operation revealed slight swelling of the optic nerve and enlargement of the median ventricle. Subsequent craniectomy in the posterior fossa disclosed an acoustic neurinoma. In another case reported by the authors, primary bilateral optic atrophy was revealed at autopsy to be due to compression of the optic nerve by an atheromatous plaque at the bifurcation of the carotid artery.

BERENS, New York. [ARCH. NEUROL. & PSYCHIAT.]

Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

GEORGE T. JORDAN, M.D., *President*

WALTER H. THEOBALD, M.D., *Secretary*

Regular Monthly Meeting, April 1, 1940

Diagnosis and Diseases of the Salivary Glands. DR. R. BRUCE MALCOLM.

Diseases of the salivary glands are unique. This illustrated talk is based on 167 cases encountered during the past seven years, in each of which a surgical operation was done. Each gross specimen was seen and examined histologically. The lesions were grouped as follows:

Parotid:		
	Acute suppurative parotitis	8
	Incised wound with paralysis of the seventh nerve	1
	Papillary cystadenoma	3
	Tuberculosis in the parotid gland	1
	Hodgkin's disease in the parotid gland	1
	Lithiasis with chronic inflammation	3
	Mixed tumors	45
	Carcinoma	23
		85
Submaxillary:		
	Chronic obstruction of the submaxillary ducts	2
	Lithiasis	5
	Aberrant salivary gland in thyroglossal duct cyst	1
	Cysts	3
	Mixed tumor	6
	Carcinoma	2
	Resection for carcinoma of the lip	55
	Resection for malignant carcinoma	3
		77
Sublingual:		
	Cysts	3
	Lithiasis	2
		5

Acute suppurative parotitis, while not common, is extremely painful and the affected glands must be drained. It may be caused by ordinary lithiasis. In 1 case listed, paralysis of the seventh nerve was due to a knife wound; the nerves were sutured with a satisfactory result. Papillary cystadenoma usually occurs as a small tumor, not nodular, of fairly rapid growth, and may undergo malignant change. One lesion diagnosed as such proved to be tuberculosis of the gland, while another was due to Hodgkin's disease.

Mixed tumors are among the great bugbears of surgical practice. At first there is simple hyperplasia, then fibroblastic hyperplasia; then the growth becomes anaplastic and one may find chondroblasts and even osteoblasts. The tumor usually appears in the lower pole as a small, gradually growing nodule. Sudden enlargement may take place after a number of years, and then the growth must be considered malignant. One case cited was that of a young man who for years had had a tumor mass the size of a walnut, which suddenly increased in size. Prior to that time he had had twenty roentgen treatments. Recently facial paralysis had occurred, with intense pain. When this triad, pain, rapid growth and facial paralysis, is present, the diagnosis of carcinoma is confirmed. Of 23 carcinomas, 7 developed in ordinary benign mixed tumors. Probably 1 in 4 will undergo malignant change, particularly if there has been partial removal and roentgen therapy. Squamous cell carcinoma may arise from such a tumor, and 6 of such origin are listed in this series. Ten were of the adenocarcinomatous type. In the case of a girl aged 5 with a tumor of the parotid gland, biopsy showed rapidly developing adenocarcinoma. An attempt was made to remove the whole tumor, but the growth was adherent to and had invaded the jaw. It was necessary to remove the jugular vein, tie off the external carotid artery and remove the entire gland.

Another patient, aged 23, had a tumor mass in the floor of the mouth. A surgeon attempted to remove it but partially removed the submaxillary duct with its nippled orifice, causing a recurring obstruction. Eventually it was necessary to do a complete extirpation of the gland. In a woman aged 20 it was necessary to cut the hyoid bone and remove a mixed tumor of the salivary gland. Carcinoma in the submaxillary glands is rare, although 2 were found in this series. Metastases in the lymph nodes necessitate removal of the submaxillary gland. In 5 cases there was secondary involvement spreading into the submaxillary gland, which seems to be resistant to squamous cell carcinoma, as in only 3 of 55 excisions of carcinoma of the lip was involvement of the salivary gland revealed.

The most malignant tumor is malignant melanoma, which almost completely destroys the salivary gland. This occurred in 3 cases. Diagnosis of all these tumors is usually made by palpation on the outside and through the mouth, which gives more information than any other procedure.

Electrocoagulation. Dr. Joseph F. Jaros.

The colored motion picture presents the actual process of electrocoagulation within body cavities such as the larynx, the rectum and the vagina. Pictures taken at a distance of 4 feet (122 cm.) from the patient, through special light projection, show, greatly enlarged, the contents only of the cavities—tonsils, hemorrhoids and uterine cervix—and suggest means of eliminating the most common objections to electrocoagulation, i.e., operative and postoperative pain, hemorrhage and scarring.

(A film was shown.)

The film presents for the first time:

1. A new principle for the elimination of hemorrhage after electrocoagulation, based on the first histomechanical principle of Thoma and the researches on capillaries by Elliot R. Clark.

2. The presence of an anarterial area of the tonsil based on the research of Fetleroff and on over 800 cases in which tonsillar coagulation was done by me.

3. The regeneration of glands and even of ciliated epithelium of the nasal turbinates and of the uterine cervix following severe electrocoagulation.

4. A partial analgesia of three or four days following the topical application of a 5 per cent solution of diothane hydrochloride.

DISCUSSION

Dr. Frank J. Novak: The members of the Society must all admit that they have seen a beautiful demonstration of color photography of the depths of a

cavity. Photographs such as these are not easy to obtain. I hesitate to renew the controversy between those who remove tonsils surgically and those who desiccate them. The matter has been discussed many times, and I have not changed my opinion. I must say, after observing a number of the patients treated by Dr. Jaros and examining them with a critical—perhaps hypercritical—eye, that I found no evidence of any retained or remaining lymphoid tissue in the fossae. The pillars were perfectly pliable, and there was no evidence after a long period of any recurrence or creeping of adjacent lymphoid tissue into the tonsillar fossa.

I am eager to hear Dr. Rukstinat speak on some phases which will be of interest to you, and I should like to yield whatever time I have left to him.

Dr. George J. Rukstinat: A microscopic study of 6 portions of turbinate and 2 uterine cervixes subjected to electrocoagulation by Dr. Jaros and later surgically excised disclosed interesting late results in tissue regeneration. The turbinates had been treated from six weeks to one and one-half years prior to their excision. Each was covered by epithelium two or more layers thick, in some places stratified squamous in nature but for the most part cuboidal. In a few of the small depressions, ciliated epithelium was found. Variability in the type of covering epithelium depends in some measure on the exposure of the turbinates to aeration. The deeper structures, such as glands and blood vessels, were, however, uniformly well preserved. Their number and distribution were normal and did not suggest prior mechanical or thermal treatment. Although the number of tissues studied was small, Dr. Jaros and I realized that a preliminary report of the favorable findings would stimulate further investigation.

Dr. Thomas C. Galloway: I was much interested in this subject at one time, and about ten years ago I tried all electrosurgical technics in about 100 cases. I concluded that electrosurgical procedure was of advantage in only a few special cases or in those in which there were flat fragments in an open fossa. Going back this year to one of the instrument houses which used to hold crowded clinics to teach how to take out tonsils by coagulation, I found the place almost deserted. I asked one of the salesmen about it, and he said—"Coagulation ain't the panacea it once was—short wave is the stuff now."

In spite of what has been said, probably the loss of time, the total disability and the pain are considerably greater in coagulation than with proper dissection. I have seen Dr. Jaros work, and there is no doubt that he exercises as much skill and care as one who does good surgical tonsillectomy. It is still my opinion, however, that of hundreds of patients I have seen whose tonsils were originally treated by electrocoagulation, there were not 6 whom I could consider to have had good clean tonsillectomies.

Dr. Robert H. Good: I think surgical diathermy is one of the most wonderful assets I have encountered in the last ten years, but I thought the matter of dissecting tonsils had been discarded so far as attempting to remove the whole tonsil with diathermy is concerned. My associates and I tried this years ago, and I could not get patients to come back ten or twenty times; many did not have the treatment completed. Another objection was that I never could tell where the limit of the capsule was. If the diathermy went too deep, there was danger of destruction of tissue. It is fortunate that electrocoagulation of the tonsils is a painful procedure, and I am sorry a new anesthesia has been found which eliminates pain for four days after the procedure.

Dr. Austin A. Hayden: I agree fully with what Dr. Galloway has said about this procedure. My experience with electrocoagulation of the tonsils has been entirely that of an observer. Briefly, the results in the patients I have seen have been terrible.

The pictures shown, taken six months or six weeks after operation, seemed to demonstrate clearly the absence of scar tissue and the complete pliability and even elasticity of the tissue of the pillars. I was glad to see these pictures, and I marvel at the ability of the doctor to make photographs inside the throat. They are the best I have seen. I commend his photography, but I distrust his surgical method.

DR. JOSEPH C. BECK: Since Dr. Jaros referred to me as one who had discarded the method of electrocoagulation, I wish to reply that the statement is not entirely correct. I have stated distinctly many times that I employ electrocoagulation for the removal of remnants of tonsils or of recurrent tissue at the base of the tongue, which I or my associates have left in the throat. When recurrences and infection have occurred in such tissue, I have felt it better to destroy the tissue by electrocoagulation than to dissect these stumps, which were in most instances not actual tonsil tissue. I have never felt that I wanted to remove tonsils by the method of electrocoagulation in contradistinction to the usual surgical method.

It was interesting from a pathologic standpoint to see the section made after electrocoagulation in which the epithelium was not only left intact but had apparently reformed. The late Dr. Bloodgood showed the use of the electrocoagulating knife with which the skin is incised and showed that primary union occurred after such incision. It must be a special type of current which permits primary union without stitch abscess or suppuration following electrosurgical incisions.

I hate to put my opinion against that of a pathologist, but one section shown tonight looked to be so poorly stained as to be atrophic. The fact that a few glands are left is no reason to say that tissue is normal. I think it was a pretty good example of coagulation having destroyed the good staining tissue.

In conclusion, I wish to say that when any method is brought up and the attempt is made to show real work, as was done tonight, physicians should be open minded and say it is good. These pictures were good; the color photography, aside from the excess of high lights, was excellent. The pliability of tissues after operation was certainly shown in these pictures; there is no question about that. There are far more mobility and less scarification than are usually seen after ordinarily performed tonsillectomy.

MR. HOWARD A. CARTER: The Council on Physical Therapy feels that the regular surgical removal of tonsils is the method of choice. For patients with serious cardiac disease, severe toxic goiter or tuberculosis in an advanced stage or when other complications make it inadvisable to use a general anesthetic, electrocoagulation is indicated.

DR. ELMER KENYON: Many years ago Dr. G. W. Hudson Makuen expressed much concern about the desirability of tonsillectomy because of the frequently resulting impairment of the tissues of the fauces and the action of these tissues after operation. He said: "You are making it impossible for children to develop singing voices." Tonsillectomy is recommended for many patients who should not be subjected to this operation. In cases in which the palate is short, if through tonsillectomy the structure is shortened still more, which is almost inevitable, a nasal voice may result.

I used to say years ago that there should be two methods of approaching the tonsil: one, removal by tonsillectomy extracapsularly, and the other, removal of the lymphatic tissue within the capsule. Operation within the capsule is not attended with deformity. My thought and experience have been to extirpate the tonsillar tissue within the capsule with biting forceps. If one could add cauterization as a final step to this procedure, such an intracapsular operation should be an excellent one.

DR. L. Z. FISHMAN: I should like to emphasize the remarks of Dr. Kenyon with respect to the need for preventing even the slightest disturbance in the motility of the posterior pillar. Not only are the structures of the soft palate concerned with resonance and articulate speech—and these represent only gross acts of the palate—but they are also great factors in the quality of the singing voice. The fact that tonsillectomy can cause a derangement in the voice by disturbing the elasticity and motility of the posterior pillar is particularly evident in an unusual specimen which I encountered in my dissections of larynxes. In this larynx, the pharyngopalatine muscle (posterior pillar) was found to be a direct combination of a number of muscle fascicles of the vocal (lateral cricoarytenoid) muscle itself.

Obviously, in this instance the posterior pillar was an integral part of the laryngeal adductors. Certainly the greatest care must be exercised in performing tonsillectomies on children who may become singers. In vocalists this operation should be avoided to the greatest extent possible; any substitute for a complete surgical tonsillectomy is to be desired in such instances.

DR. WALTER THEOBALD: Some few years ago, you will recall, the subject of electrocoagulation was presented before this Society, and I think at that time it was pretty well hashed over and closed. Some 12 patients whose tonsils had been removed by this method were presented from the clinic of Rush Medical College, and in not 1 was the result satisfactory. That was the admission of the operators as well as of the observers. Dr. Beck has proposed that these patients of Dr. Jaros be brought in for observation. This presentation was given primarily to demonstrate some fine photography. Knowing full well that there would be a stiff argument condemning the electrosurgical procedure, Dr. Novak and I thought the work was worthy of presentation because of the excellent color photography of the inside of a cavity. If it is the wish that this report be brought up at some future time, I suggest that a small group be detailed to examine some of Dr. Jaros' patients and report to the Society.

DR. JOSEPH F. JAROS: I wish to thank the Society for its kind reception of this presentation. Dr. Good directed attention to the striking elasticity of the pillars following electrocoagulation. This lack of scarring is especially characteristic after the use of the multistage method and is particularly desirable, as Dr. Kenyon clearly brought out in his discussion, in the supratonsillar fossae and in the posterior pillars. This feature alone is worthy of further study.

Dr. Novak and Dr. Galloway have kindly corroborated the film demonstration of possible satisfactory results. This possibility can be more frequent as pain is more and more eliminated, since absence of pain insures more thorough and more rapid work.

The absence of recurrence is interesting and although, as Dr. Galloway has stated, complete removal of tonsils may be more difficult by electrocoagulation than by surgical procedure, recurrence and regrowth have not been observed in the many patients reexamined over a ten year period among more than 800 who were treated by this method. Removal has been satisfactory in cases of angina pectoris and coronary thrombosis.

The suggestion that patients on whom the procedure has been completed might be presented before a committee is well taken. Fully 24 patients treated during the past ten years have agreed to submit to inspection.

Physiology of Mastication and Deglutition. ALLAN BRODIE.

Hearing Aids—Tube or Carbon? DR. AUSTIN A. HAYDEN.†

At present both tube and carbon aids have definite advantages and disadvantages. Both are being improved constantly.

Indications for each must be formulated from the experience of otologists, physicists and manufacturers, with the final decision being made by the patient.

Tube aids give more brilliant hearing improvement in conduction loss when nerve function is good. In severe perceptive loss, results are often disappointing.

Purchase from a firm that manufactures both carbon and tube aids, for air as well as for bone conduction, allows choice between the two and makes exchange of one for the other easier.

Since tube aids are much more complex instruments, the need for an impartial evaluating agency such as the Council on Physical Therapy of the American Medical Association, to set and maintain requirements for acceptance of standards of construction and performance as well as of sales ethics and advertising, is even more necessary with the advent of tube aids. Financial rating of the manufacturers is very important.

† Dr. Hayden died July 10, 1940.

Although the lure of radio tubes is great, patients should not discard carbon for tube aids without careful investigation.

Tube aids are as yet more expensive to manufacture, buy, keep up and service.

Whether tube aids will supplant carbon aids or vice versa, or whether each will eventually occupy its own definite field, the actual improvements that are being made in construction by manufacturers and in the prescription of hearing aids by otologists practically assure good results for the vast majority of patients properly fitted with a well made hearing aid.

Even better instruments and better hearing seem to be on the way.

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DISCUSSION

MR. HOWARD A. CARTER: (Demonstration with slides.) I shall not take your time to discuss the old mechanical aids, such as rubber tubes, ear trumpets and fans. The first slide shows a schematic diagram of the carbon granule transmitter hearing aid. This hearing aid is of a circuit similar to that used by the one way telephone and operates as follows: The sound energy impinging on the diaphragm sets it in motion. This changes the pressure on the carbon granules, thereby varying the resistance in the circuit. The energy from the battery is varied in the entire circuit, which passes through to the receiver. Varying current in this circuit changes the magnetic flux in the permanent magnet of the receiver and hence sets its diaphragm in motion. This in turn sets up a sound disturbance in the vicinity of the deafened ear.

The vacuum tube aid, as may be observed from the schematic diagram in slide 2, is somewhat more complicated. In the vacuum tube aid the crystal microphone is used. This crystal is a miniature generator developing a feeble electric current, which is amplified by three vacuum tubes. The energy for amplification comes from the A and B batteries. Finally, the energy which has been amplified is delivered to the receiver, which may be of either a crystal or an electromagnetic type.

DR. G. H. MUNDT: I think many people share my predicament. I never know when to prescribe a hearing aid. I have read and heard a great deal about it, but I have never been quite clear as to whether a hearing aid should be prescribed for the person who has lost a little hearing or whether one should wait until he is entirely deaf, or whether a hearing aid should be prescribed at all or lip reading recommended. I wish Dr. Hayden would give a word on that.

DR. ROBERT H. GOOD: Would a wet battery produce less noise, and would the tubes last longer with a wet battery than with a dry battery? I know the wet battery gives more smooth results. In dry batteries air bubbles form and change the voltage, which causes noise.

MR. HOWARD A. CARTER: By wet batteries I understand you to mean storage batteries. The answer is that there should be no significant difference. It is assumed that "static" means the internal noises in the hearing aid. I am not quite clear as to why wet batteries should give more smooth results, as you say. I should think a dry battery made up of several cells would give as smooth if not smoother results than a small storage battery. It is possible to charge a storage battery over and over. The lasting qualities of the hearing aid should not be any different whether a storage battery or a dry battery is used.

DR. L. J. HUGHES: I think the sooner a deaf person becomes adapted to a hearing device the better. It helps him to retain his natural voice, and a high nasal voice does not develop.

DR. AUSTIN A. HAYDEN: The question that Dr. Mundt brings up is important. My answer is to invite him to the meeting of the Illinois State Medical Society, where the subject "For Whom Should Hearing Aids Be Prescribed?" is to be dealt with. In general, hearing aids should be prescribed for people whose hearing is defective and can be improved by an aid. There are many causes for exceptions

to that—the psychologic factors, the matter of expense and the matter of pride. People do not want to wear these devices. Lip reading is regarded as a natural human hearing aid, and therefore lip reading and the use of hearing aids should go together. Lip reading is an art—an inexact art. It cannot be used unless strict attention is paid to the context of what is being spoken. Many sounds are formed in the same way by the lips, and the context determines what they mean.

In reply to Dr. Good's question about wet batteries (the small type operated with a few drops of fluid), that subject has been gone into extensively by the companies which manufacture hearing aids. Their experience and that of patients has not been such as to make the use of this type of battery acceptable. Only one company pushes the use of wet batteries, and all in all the sales of this type have been a very small percentage. The expense connected with the battery of a hearing aid is $2.50 a month, which could be reduced considerably. It is astonishing that it is necessary to use only two batteries, alternating day by day, and to instil 2 drops of water into each after it has been used. That simple direction is seldom followed. I have no evidence whether wet batteries are harder on tubes than dry cells or whether there is less extraneous noise.

With reference to Dr. Hughes's statement, the evidence is not at all conclusive. The natural voice depends on the retention of bone conduction, and whether or not a hearing aid, even though worn as continuously as possible, will actually preserve the voice is something which is disputed. I know Dr. Hughes's experience and respect that highly, but there is much evidence contrary to his opinion.

Directory of Otolaryngologic Societies *

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. LeRoy A. Schall, 243 Charles St., Boston.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia.
Place: Cleveland. Time: June 2-6, 1941.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Frank Brawley, 30 N. Michigan Ave., Chicago.
President-Elect: Dr. Frank R. Spencer, Physicians' Bldg., Boulder, Colo.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Chicago. Time: Oct. 19-24, 1941.

American Broncho-Esophagological Association

President: Dr. Gabriel Tucker, 250 S. 18th St., Philadelphia.
Secretary: Dr. Paul Holinger, 1150 N. State St., Chicago.
Place: Cleveland. Time: June 3, 1941.

American Laryngological Association

President: Dr. Gordon Berry, 36 Pleasant St., Worcester, Mass.
Secretary: Dr. Charles J. Imperatori, 108 E. 38th St., New York.
Place: Atlantic City, N. J. Time: May 28-30, 1941.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. J. Mackenzie Brown, 1136 W. 6th St., Los Angeles.
President-Elect: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.
Place: Ambassador Hotel, Los Angeles. Time: June 16-18, 1941.

SECTIONS:

Eastern.—Chairman: Dr. N. S. Weinberger, Robert Packer Hosp., Sayre, Pa.

Southern.—Chairman: Dr. William G. Kennon, Doctors Bldg., Nashville, Tenn.

Middle.—Chairman: Dr. Walter H. Theobald, 307 N. Michigan Ave., Chicago.

Western.—Chairman: Dr. Robert C. Martin, 384 Post St., San Francisco.

American Otological Society

President: Dr. George M. Coates, 1721 Pine St., Philadelphia.
Secretary: Dr. Isidore Friesner, 36 E. 73d St., New York.
Place: Atlantic City, N. J. Time: May 26-27, 1941.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.